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CHANGES IN THE INTERNAL EAR DUE TO INCREASED ENDOCRANIAL PRESSURE

THE HISTOLOGIC BASIS OF CONGESTIVE INNER EAR

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The question whether changes in the inner ear similar to choked disk in the eye occur as a result of increased endocranial pressure has not yet been definitely answered. This is shown by the confusion in nomenclature seen in such terms as choked labyrinth, neuritis of the eighth nerve, stagnation dropsy, neuritis labyrinthica and congestive inner ear.[1] Furthermore, many otologists deny absolutely that such changes take place.

Likewise the question of endocranial pressure itself is still in dispute. It is known to arise as a result of the disproportion between the size of the cranial cavity and its contents. A differentiation is made according to whether the pressure increases rapidly, as in acute processes, or gradually, as in chronic conditions. In this paper I shall deal only with chronic brain pressure. If one studies the enormous literature covering endocranial pressure, one finds that one can classify the various theories, according to the three main substances in the area, cerebrospinal fluid, blood and brain tissue, into three groups. One group explains the changes in pressure as caused by the cerebrospinal fluid; another attributes them to circulatory disturbances, while the third believes that direct compression of the brain substance is the cause.

How does chronic endocranial pressure affect the ear? Let me differentiate between the clinical symptomatology and the pathologic anatomy.

The earlier literature deals largely with the clinical side. Thus, for example, I [2] myself twenty years ago wrote an article entitled "Tumor

1. Moos, S.: Arch. f. Augen- u. Ohrenh. **4**:179, 1874. Panse, R.: Arch. f. Ohrenh. **70**:15, 1906. Cushing, H. W.: Tumors of the Nervus Acusticus and the Syndrome of the Cerebellopontile Angle, Philadelphia, W. B. Saunders Company, 1917. Beck, J., cited by Brunner, H.: Otologische Diagnostik der Hirntumoren, Berlin, Urban & Schwarzenberg, 1936, p. 3. Habermann, J.: Ztschr. f. Ohrenh. **75**:19, 1917. Wittmaack, K.: Arch. f. Ohren-, Nasen- u. Kehlkopfh. **99**:71, 1916.

2. Fischer, J.: Verhandl. d. Gesellsch. deutsch. Hals-, Nasen- u. Ohrenärzte **1**:297, 1920; Monatschr. f. Ohrenh. **55**:371 and 531, 1921.

of the Brain and the Ear," in which I reported a study of 130 cases of neoplasm of the brain observed in the surgical clinic of the University of Vienna. The clinical symptoms of congestive inner ear were described in that paper as follows: subjective noises in the head, tinnitus, diminished hearing, shortened bone conduction, lowered upper tone limit, dizziness, spontaneous nystagmus, abnormal irritability of the labyrinths, disproportion between objective labyrinthine reaction and subjective sensation of dizziness and, finally, marked changes in symptoms on repeated examinations. These symptoms of congestive inner ear were described as occurring in 77 per cent of cases of tumors of the brain in the anterior and the middle fossa, in comparison with choked disk in the eye, found in 90 per cent of the same cases.

Of much greater significance is the work dealing with the anatomic changes in the inner ear due to chronic brain pressure. A study of the literature, however, revealed the astounding fact, that, despite the importance of this subject, only a few positive findings are available. The reason for this is that the examinations were made on unsuitable material. Almost all the investigators described cases of tumor of the acoustic nerve or some other neoplasm of the posterior fossa; it was, therefore, impossible to decide whether the changes described in the inner ear were due to the chronic brain pressure (congestive inner ear) or the result of local damage.

In 1925 I [3] directed attention to this point, when I began the study of the histologic basis of congestive inner ear, on extensive autopsy material. In order to rule out local damage, I suggested strict requirements as follows: All cases of tumor of the acoustic nerve, cerebellar tumor or tumor of the pontile angle, in which the growth has encroached on the internal meatus so that it damaged the cochlear nerve directly were to be excluded. In the same manner must be excluded all cases of tumor of the posterior fossa in the vicinity of the petrous bone and all cases in which only the diseased petrous bone was microscopically examined. Naturally operative procedures, such as extirpation and decompression, may influence the histologic structure, because of hemorrhages and damage to the brain tissue.

Examining the literature with this point in view, I found only 1 case which fulfilled all the requirements. This was a case reported by Steinbrügge [4] in 1891. He investigated a tumor of the corpora quadrigemina with accompanying hydrocephalus, with which he found a depression of Reissner's membrane and bulging of the secondary tympanic membrane.

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3. Fischer, J.: Allgemeinerscheinungen im Bereiche des Cochlearapparatus, in Alexander, G., and Marburg, O.: Handbuch der Neurologie des Ohres, Berlin, Urban & Schwarzenberg, 1925, vol. 2, p. 1521.

4. Steinbrügge, H.: Pathologische Anatomie des Gehörorganes, in Orth, J.: Lehrbuch der speciellen pathologischen Anatomie, Berlin, A. Hirschwald, 1891, p. 119.

My material in 1925 was obtained at autopsy from 5 patients who had died of neoplasms of the brain in either the anterior or the middle fossa and who had had no surgical intervention. Complete bilateral serial sections (vertical to the petrous bone) were made.

Studies on these sections of the 10 petrous bones revealed numerous changes which were briefly summarized as follows:

1. Transudations and exudations (in the scala tympani and scala vestibuli, in the ductus cochlearis, in the aqueducts and also perineurally).

2. Hyperemia (especially venous, in the soft parts of the inner ear and of the petrous bone).

3. Hemorrhage (recent and old, in the membranous inner ear and in the labyrinthine capsule).

4. Changes in the walls of the blood vessels.

5. Local congestions (as in the nerve, the spiral ligament or the modiolus).

6. Cellular infiltrations.

7. Pigmentations.

8. Adhesions (in the membranous inner ear).

9. Formative changes of the membranous inner ear (ectasia, stenosis and collapse).

10. Atrophic and degenerative changes (in the sense organ and in the nerve ganglion apparatus).

11. Osseous changes (pressure atrophy).

For details and photomicrographs of the histologic changes the reader is referred to the original article.[5] The findings were confirmed by some but opposed by others.

G. Alexander,[5] in his address before the American College of Surgeons at Detroit in 1927, reviewed the findings and confirmed them, using the original photomicrographs.

S. J. Crowe[6] reported in 1929 on 2 neoplasms of the brain with changes in the inner ear. Although the histologic slides demonstrated the pathologic changes excellently, 1 patient had had chronic otitis media since childhood and did not have choked disks, and the other patient had a cerebellar tumor. Thus, local changes could not be excluded.

After that, attempts were made to solve the question of congestive inner ear experimentally. The first investigator (Marx[7]) found no

5. Alexander, G.: Surg., Gynec. & Obst. **46**:361, 1928.

6. Crowe, S. J.: Anatomic Changes in the Labyrinth Secondary to Cerebellopontile and Brain Stem Tumors, Arch. Surg. **18**:982 (April) 1929.

7. Marx, H.: Ztschr. f. Hals-, Nasen- u. Ohrenh. **9**:135, 1924.

changes in the inner ear, but later experiments (Dunkel,[8] Tobeck[9]) were successful.

Dunkel[8] produced increased intracranial pressure in animals by the following procedures:

1. Forcible injections of Ringer's and Locke's solutions in the subdural spaces repeated over a long period.
2. Endocranial injections of paraffin.
3. Injections of paraffin into the aquaeductus Sylvii.
4. Injections of emulsion of lamp black into the subarachnoid spaces.

These experiments produced the same histopathologic changes of the inner ear as I found in man.

Tobeck[9] injected a large amount of paraffin and normacol[9a] in the regions of the internal auditory meatus. The animals given the injections lived from two days to twenty-one weeks. His findings were as follows: The changes in the inner ear were marked with acute endocranial pressure, but not so marked or absent with chronic pressure, the increased endocranial pressure being transmitted through the blood circulation. He denied any analogy between choked disk and congestive inner ear because of anatomic differences and because of the transmission of the pressure by means of the circulating system.

I find myself unable to agree with the conclusions drawn by Tobeck, for the following reasons:

(*a*) With sudden injections of such a large amount of paraffin local damage cannot be excluded.

(*b*) With injections in the region of the internal acoustic meatus local injuries may occur.

(*c*) The length of time that most of the animals lived after the injections was too short to establish chronic brain pressure.

In an article[10] written by me in 1935, entitled "Meningioma of the Posterior Fossa," the statement was made that the changes in the inner ear are due to local brain pressure accompanying such conditions as multiple hernias of the brain and vaginal dropsy of the ninth nerve (as described by Brunner[11] affecting the eighth nerve in a case of tumor of that nerve). The present study shows the same findings with neoplasms of the anterior and the middle fossa, so that the conclusion now drawn is that chronic brain pressure played a role in their production also.

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8. Dunkel, G.: Folia neuropath. eston. **8**:1, 1928.

9. Tobeck: Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres **30**:341, 1933.

9a. Normacol is a proprietary preparation usually employed as a laxative. It is a plant mucus of the bassorin series, with the addition of Rhamnus frangula.

10. Fischer, J.: Monatschr. f. Ohrenh. **69**:40, 1935.

11. Brunner, H.: (*a*) Ztschr. f. d. ges. Neurol. u. Psychiat. **132**:57, 1931; (*b*) Ztschr. f. Hals-, Nasen- u. Ohrenh. **30**:443, 1932.

In 1936 H. Brunner,[12] of Vienna, in his excellent book stated that the question of the congestive inner ear has not been as yet definitely decided and that further work is necessary to clear up the point. He also made histopathologic examinations. His conclusion was that only with acute changes of the brain pressure are there marked changes in the inner ear but that with chronic generalized brain pressure such changes are lacking. He refused to accept the analogy between choked disk and congestive inner ear, also because of anatomic considerations.

If one is not content with Brunner's conclusions and refers back to the histopathologic pictures described, one will see that many changes in the inner ear are present. Brunner merely differs in interpretation. The changes seen are explained by him as due to various causes, such as postmortem alterations, artefects, inflammations, congenital hypoplasia, physiologic variations and senility. He attributed only two changes (hernia of the brain and edema of the ductus endolymphaticus) to generalized brain pressure. The justification of the analogy between choked disk and congestive inner ear will be discussed later in this paper.

In the most recent article on this subject, B. Hansson and C. O. Nylén [13] described 6 cases of tumor of the brain. They stated that only postmortem and no pathologic changes were observed in the inner ear. But in reviewing the description of their material one finds that they admit that most of the cochlea dropped out of the preparation, was destroyed by postmortem changes or suffered, in any case, because of unsatisfactory fixation, so that it is not clear how the authors could conclude that no pathologic changes were present.

Regarding the study of the membranous inner ear in general, this is a good opportunity to stress the need for ideal fixation of the petrous bone, if the histopathologic picture of the fine structure of the sense organ and the nerve ganglion apparatus is to be investigated. With such technic the literature would less often reveal reports stating that only postmortem and no pathologic changes are present. In any case, it is clear that the changes observed can be normal, postmortem or pathologic and that postmortem changes often destroy evidence of pathologic changes.

From the preceding references, it is clear that there has not as yet been a universal acceptance of the anatomic basis for congestive inner ear. Therefore, I felt that more microscopic study of clinical material was necessary. Accordingly I examined within the last few years specimens from hundreds of cases of neoplasms of the brain. From these I was able to select a new group of 5 in which the patient had died of tumor of the anterior and the middle fossa and had not had

12. Brunner, H.: Otologische Diagnostik der Hirntumoren, Berlin, Urban & Schwarzenberg, 1936.

13. Hansson, B., and Nylén, C. O.: Acta oto-laryng. **23**:370, 1936.

any surgical intervention. Thus serial sections of 10 petrous bones were obtained, which were stained and sectioned with the same technic as that used in the earlier series of 10 discussed in my 1925 article.

For clarity of presentation, excerpts of the hospital records will be given first, then the clinical otologic findings, the histologic description of the petrous bones and finally brief comment on each case. The cases are arranged according to the degree of change found, the specimens from the first case showing the early stage of congestive inner ear and those from each succeeding one showing more advanced changes than those from the preceding.

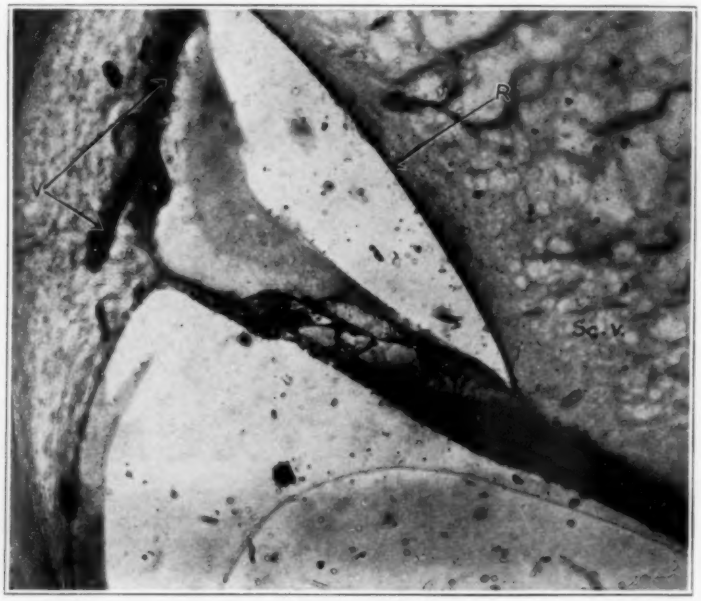

Fig. 1.—Photomicrograph from a case of frontal tumor; vertical section through the cochlea, showing the scala vestibuli (*Sc.v.*) and the ductus cochlearis filled with transudate (*t.*). Reissner's membrane (*R.*) is bulged outward. There are engorged vessels (*v.*) in the stria vascularis and in the ligamentum spirale, which is also edematously infiltrated. Although embedded in transudate the organ of Corti is intact.

REPORT OF CASES

CASE 1.—A 32 year old white man was admitted to the hospital with headache, weakness, anorexia, tinnitus (chirping and twittering), a sensation of fulness and seizures of jacksonian epilepsy. On examination the following signs were found: pain on percussion over the anterior part of right side of the cranium, left hemiplegia, paralysis of the left facial nerve, deviation of tongue to the left, a positive Babinski sign and choked disks.

Roentgen examination revealed deep impressiones digitatae in the frontal and parietal regions.

Otologic Findings.—Both drum membranes appeared normal. Perception of the accentuated whispered voice was 20/20 in each ear and that of the tuning forks C and c-4 normal, but the bone conduction was slightly shortened (ten seconds less than normal). Nystagmus was seen only in the end position. While the reaction to bilateral calorization was negative, that to unilateral calorization showed hyperirritability of each labyrinth. The falling and past pointing reactions were typically normal.

Autopsy.—The patient died six weeks after admission.

The autopsy was reported as revealing endothelioma of the right motor region.

Histologic Observations on the Right Petrous Bone: The labyrinthine capsule was markedly congested; the internal meatus was not enlarged. The cerebral ostium of the aquaeductus cochleae also showed signs of congestion in the form of

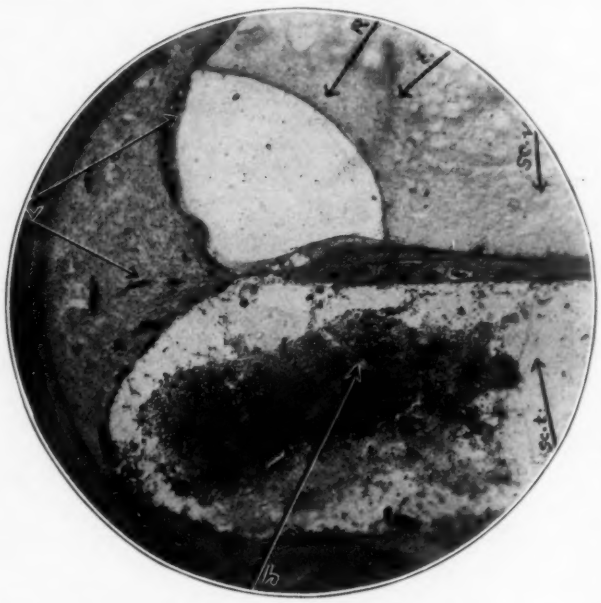

Fig. 2.—Photomicrograph from a case of frontal tumor; vertical section through the cochlea revealing the scala vestibuli (*Sc.v.*) filled with transudate (*t.*) and recent extensive hemorrhage (*h.*) in the scala tympani (*Sc.t.*). The marked bulging of Reissner's membrane (*R.*) has resulted in ectasia of the ductus cochlearis. Transudate (*t.*) surrounds the organ of Corti. Engorged vessels (*v.*) are seen in the stria vascularis and the spiral ligament.

subepithelial edema of the wall and transudation and recent hemorrhages in the lumen. The saccus endolymphaticus was occluded by subepithelial edema with resultant bulging of the endolymphatic duct above the occlusion, which was filled with homogeneous pinkish fluid.

Membranous Inner Ear and Nerve Ganglion Apparatus: A transudate was found in the scala of the cochlea and a slight amount in the ductus cochlearis of the basal turn. There was ectasia of Reissner's membrane and transudate in the tunnel space. The papilla basilaris, although embedded in the transudate, showed no changes. (The hair cells and supporting cells were intact [fig. 1]). The stria

vascularis contained engorged vessels; the vas spirale was prominent; the spiral ligament was edematous, and it also contained markedly engorged blood vessels. The spiral ganglion and the spiral nerve were normal. In the stem of the cochlear nerve was perineural and perivascular transudation, which extended into the internal meatus to the lamina cribrosa and partly into the tractus spiralis as far as the spiral ganglion. Recent hemorrhages were seen in the internal meatus. Transudate was present in the perilymphatic cistern of the vestibule and on the floor of the sacculus. There was ectasia of the ductus reuniens, with some transudate in its lumen. The other parts of the petrous bone were normal.

Histologic Observations on the Left Petrous Bone: The same changes were observed as in the right, only that the hemorrhages, especially in the scala tympani of the vestibular and the basal turn, were more extensive. Also, there was more marked bulging of Reissner's membrane, with ectasia of the ductus cochlearis and the ductus reuniens (fig. 2). The papilla basilaris was likewise intact, although embedded in the transudate.

Comment.—In this case the early stages of congestive inner ear were presented. The clinical symptoms, tinnitus, sensation of fulness and shortened bone conduction, with no changes in acuity of hearing, can all be explained by the histologic observations of perineural and perivascular transudation and the dropsy in the scalae. The organ of Corti, although embedded in homogeneous fluid, remained intact. It seems reasonable that if the patient had lived longer the increased endocranial pressure would have acted on and damaged the delicate cells of the organ of Corti and a more marked clinical picture would have resulted.

Case 2.—A 41 year old white man entered the hospital with the following symptoms and signs: headache, nausea, vomiting, paresthesia, jacksonian epilepsy, episodes of sensorial aura, right hemiparesis and choked disks.

The roentgen examination showed deep impressiones digitatae suggestive of endocranial pressure.

Otologic Findings.—The otoscopic examination gave essentially normal results. The distance at which the accentuated whispered voice could be heard appeared somewhat diminished on both sides. Bone conduction was slightly shortened, the C fork being well heard, while perception of the c-4 fork was shortened, on the right ten seconds and on the left twelve seconds less than normal. There was spontaneous nystagmus of the first degree to both sides. The Romberg test gave negative results. The response to bilateral calorization was negative, while the unilateral test showed increased irritability of both labyrinths. Falling and pointing reactions were typically normal.

Autopsy.—The patient died two months after admission.

The autopsy was reported as revealing glioma of left parietal lobe, extending anteriorly into the gyrus centralis anterior.

Histologic Observations on the Right Petrous Bone: Pinkish homogeneous fluid was noted in most of the lymph spaces of the labyrinthine capsule. Close to the inferior surface of the pyramid was a large bone cell filled with transudate. On the posterior surface of the pyramid, beneath the internal meatus, the blood vessels were engorged. Congestion was marked within the modiolus and the septum

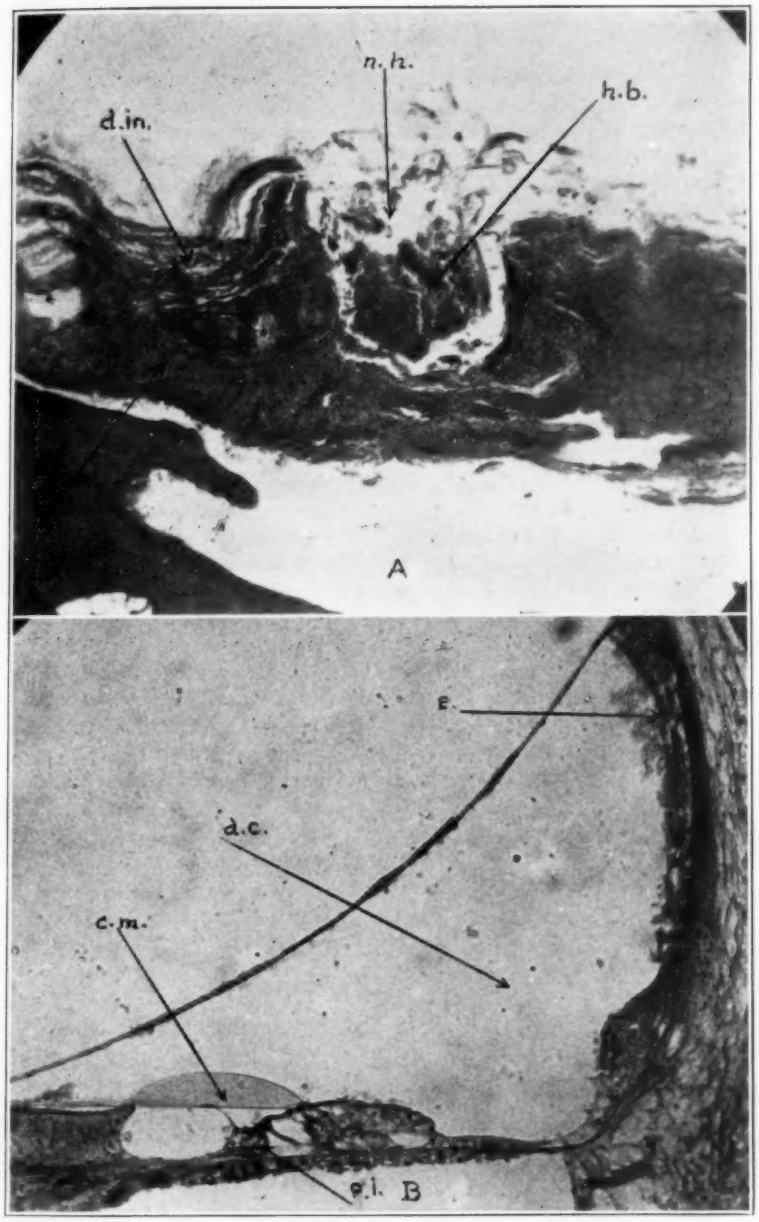

Fig. 3.—*A*, photomicrograph from a case of parietal tumor; hernia of the brain (*h.b.*) entering the dura of the middle fossa in the region of the tegmen tympani. The split dura, of which the outer layer (*d.ex.*) serves as periost and the inner layer (*d.in.*) shows a defect for the neck of the hernia (*n.h.*). *B*, photomicrograph from a case of parietal tumor; vertical section through the middle turn of the cochlea, showing moderate exudate (*E*) in the ductus cochlearis (*d.c.*), particularly on the stria vascularis and in the tunnel space. Beginning adhesions are seen between the inferior surface of Corti's membrane (*c.m.*) and the inner pillar of the papilla basilaris (*p.i.*).

scalarum. Most of the blood vessels revealed hyaline thrombosis and changes of their walls. On the anterior surface of the pyramid were multiple hernias of the brain. Above the tegmen tympani the dura was split into two layers: The outer, or inferior, layer was the periostium covering the bone of the middle fossa; the inner, or superior, layer covered the brain and revealed an opening for the entrance of a hernia of the brain (neck of the hernia [fig. 3 *A*]). Under high power magnification distinctly pyramidal cells within the herniation could be seen. Between the meshy arachnoidea and the layers of the dura was a fissure-shaped space. Another hernia of the brain appeared, lateral to this herniation but much smaller and containing hemorrhages. This one was connected with two epithelial strands of cells. A third large hernia lay above the external meatus in the middle fossa.

Membraneous Inner Ear and Nerve Ganglion Apparatus: In the vestibular turn of the cochlea was seen an exudate poor in cells, which lay on the outer surface of Reissner's membrane (perilymphatic). Reissner's membrane was bulged out. On the papilla basilaris were noted changes of the hair cells, but the supporting cells were intact. The stria vascularis was rich in blood vessels and covered by a layer of exudate. The spiral ganglion contained engorged blood vessels; the interstitial tissue was edematous, but the ganglion cells were, for the greater part, intact. In the second turn of the cochlea was seen a small amount of exudate in the sulcus spiralis internus, in the angle of insertion of Reissner's membrane in the tunnel space and between Corti's membrane and the papilla basilaris. In the latter was a beginning adhesion (fig. 3 *B*). The ciliated cells were denuded but well seen; the supporting cells were normal; the spiral ganglion was intact. In the top turn of the cochlea the ciliated cells and the supporting cells were normal; the stria vascularis showed engorged blood vessels, and the vas spirale appeared prominent. In the perilymphatic duct, close to its tympanic ostium, exudation was found. The veins of the aquaeductus cochleae were congested. There was an accumulation of fluid and congestion of the vessels in the ductus reuniens and the sacculus. Exudate with few cells was noted in the utriculus, close to the beginning of the ductus endolymphaticus, as well as transudate in the saccus endolymphaticus.

Histologic Observations on the Left Petrous Bone: The same observations as in the right were noted, but the congestion was much more marked in the blood vessels of the modiolus, of the septum scalarum and of the labyrinthine capsule. In the lymph spaces of the cochlear capsule was an accumulation of pinkish homogeneous fluid. In the endolymphatic duct subepithelial edema could be seen. Within the tissue of the hernias of the brain marked hemorrhages appeared.

Comment.—In this case the clinical symptoms of congestive inner ear were more marked than in the previous case. Besides shortened bone conduction, diminution of the hearing for the upper tone limit and slight diminution for the accentuated whispered voice occurred.

The histologic basis for these clinical symptoms can be seen in the following observations:

First, in the vestibular and the basal turn of the cochlea were exudation with degeneration of sensory cells and edema of the interstitial tissue of the spiral ganglion.

Secondly, in the second turn of the cochlea, although only a small amount of exudate was seen, were beginning organization processes causing adhesions between Corti's membrane and the inner pillar of the organ of Corti with loss of cilia.

Thirdly, there were marked congestion of blood and lymph vessels and multiple hernias of the brain on both sides, due to the increased generalized brain pressure.

CASE 3.—A 35 year old white woman was admitted to the hospital presenting headache, nausea, vomiting, dizziness, disturbances of equilibrium, ataxic gait, paralysis of the left facial nerve, diminished corneal reflexes, weakened hand grasps and choked disks.

The roentgen findings revealed increased endocranial pressure.

Otologic Findings.—First Examination: Both drum membranes appeared normal. Perception of the accentuated whispered voice was 14/20 on the right side and 12/20 on the left. Bone conduction and perception of the c-4 fork were shortened on both sides. Spontaneous nystagmus appeared in the first degree to both sides. The reaction to perception of the bilateral calorization was negative, while the unilateral revealed hyperirritability of both labyrinths. There was no spontaneous past pointing, and the falling and pointing reactions were typically normal. Diadokokinesis was present, but there were no other disturbances of coordination. The otologic findings were overwhelmingly against tumor in the posterior fossa.

Examination Three Weeks Later: No changes in hearing appeared. Spontaneous nystagmus, horizontal with a slight rotary component, of the first degree to both sides was present. The reaction to bilateral calorization was negative. Falling and pointing reactions were typically normal.

Autopsy.—The patient died five weeks after admission.

Autopsy was reported as revealing glioma of the anterior part of the left cerebral hemisphere.

Histologic Observations on the Right Petrous Bone: The blood vessels over the posterior surface of the pyramid and in the petrosal angle were markedly congested. Similar congestion, with hyaline thrombosis of the vessels, was noted in the lymph spaces of the cochlear capsule. Circumscribed enlargement of the internal auditory meatus was due to erosion of the bony wall caused by the ingrowth of pacchionian granulations. Figure 4 *A* shows distinctly the arachnoid sheath piercing the dura and eroding the bony wall in the form of pacchionian granulations. There is seen also, lying between the split dura mater, a large hernia of the brain, which is divided into four compartments by septums of connective tissue (fig. 4 *B*). Under higher power magnification brain tissue with pyramidal cells was recognized. Endothelial strands were found in the dura, which was devoid of blood vessels. There were subepithelial edema in the aquaeductus cochleae and loose areolar tissue in the perilymphatic duct. The cerebral ostium of the aquaeductus cochleae was funnel shaped in its bony portion. While the dura covered the bony canal as periostium, the arachnoid, instead of coating the glossopharyngeal nerve as a thin layer, here formed meshes or loose convolutions within the lumen. These arachnoid convolutions were edematous and showed finger-like projections (two long and three short) into the dura, as pacchionian granulations. Where the larger granulations occurred, erosions of bone were seen, an observation substantiated by the presence of osteoclasts and Howship's lacunas. Within one loop of the arachnoid convolutions were amyloid bodies.

Membranous Inner Ear and Nerve Ganglion Apparatus: The slide revealed stenosis of the ductus cochlearis in the vestibular turn of the cochlea due to adhesions between Reissner's membrane, Corti's membrane and the stria vascularis, resulting from organized exudates (fig. 5 *A*). On the papilla basilaris the cilia were absent and the ciliated cells degenerated, but the supporting cells were intact.

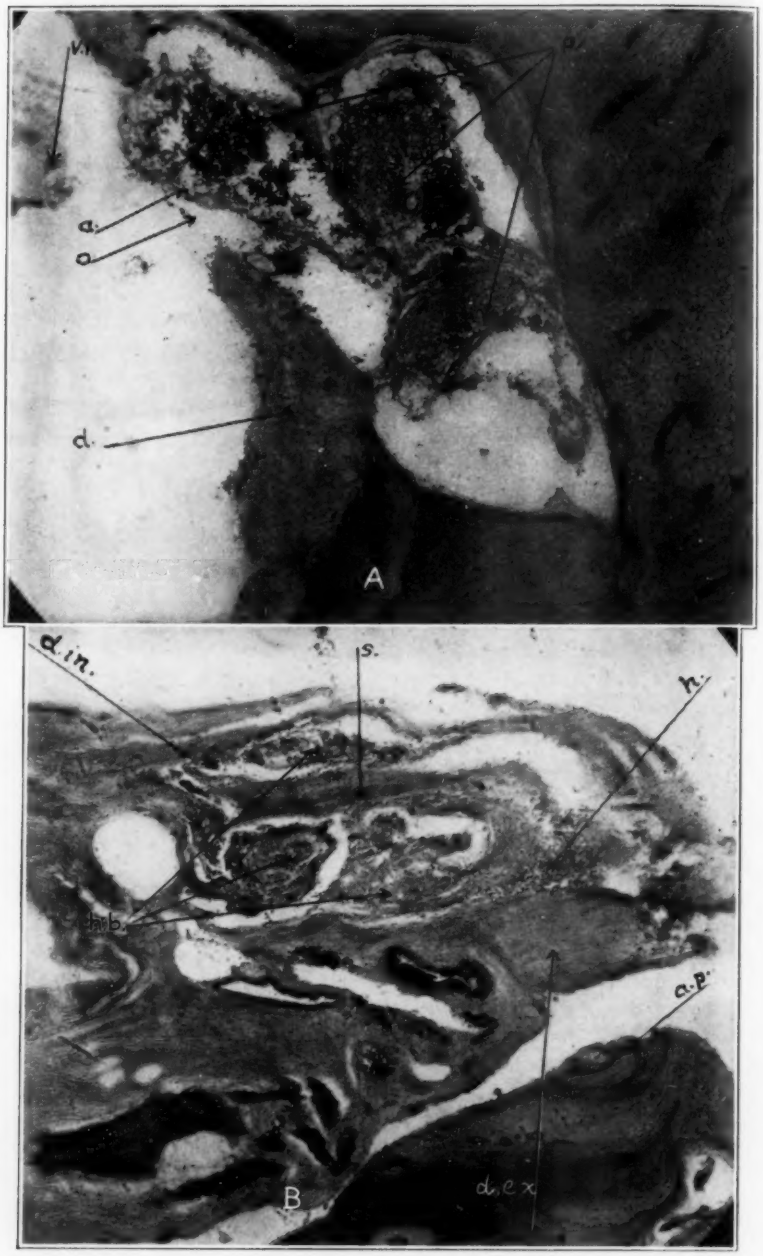

Fig. 4.—*A*, photomicrograph from a case of frontal tumor; section through the internal meatus, showing marked thickening of the dura (*d.*), which is pierced at (*o*) by the arachnoid sheath (*a.*) of the cochlear nerve (*VIII*). Pacchionian granulations (*p.*) are seen invading the bony walls of the canal. *B*, photomicrograph from a case of frontal tumor; hernia of the brain (under high power) above the anterior surface of the pyramid (*a.p.*). The dura is split into internal (*d.in.*) and external (*d.ex.*) layers, with septums of connective tissue (*s.*) separating a hernia of the brain (*h.b.*). Within the divisions pyramidal cells can be recognized. Recent hemorrhages are seen at (*h*).

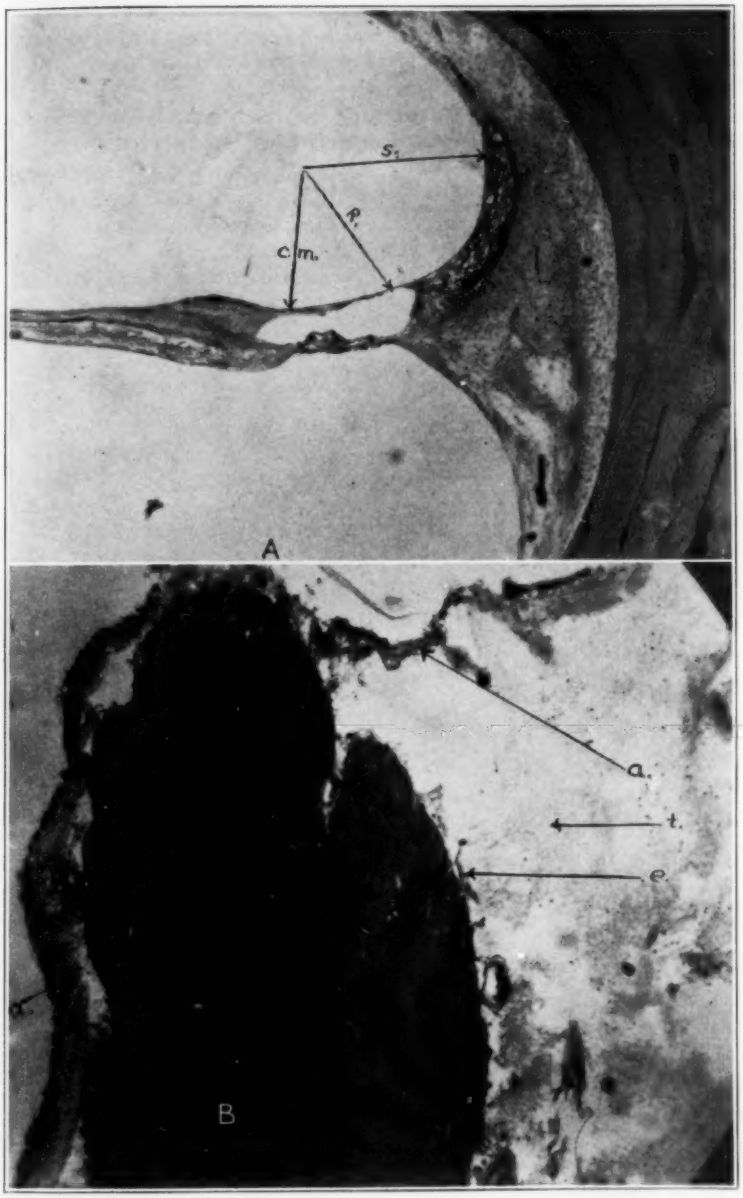

Fig. 5.—*A*, photomicrograph from a case of frontal tumor; vertical section through the vestibular turn of the cochlea, showing complete adhesion between Reissner's membrane (*R.*) and the stria vascularis (*s.*), as well as Corti's membrane (*c.m.*), with resulting stenosis of the ductus cochlearis. The sensory cells of the organ of Corti show degeneration. *B*, photomicrograph from a case of frontal tumor; vaginal dropsy of the cochlear nerve in the internal meatus. A large space filled with transudate (*t.*) lies between the epineurium (*e.*) of the eighth nerve and the infiltrated and markedly lifted arachnoid sheath (*a.*).

There was edema of the interstitial tissue, and the spiral ganglion cells were somewhat degenerated. The stria vascularis was adherent to Reissner's membrane from the upper angle of insertion to the vas prominens. Edema of the ligamentum spirale was present. The cochlear nerve within the internal meatus showed extensive vaginal dropsy. This is seen if one examines figure 5 *B*, in which one can easily note a large space between the markedly lifted arachnoid and the epineurial sheath of the cochlear nerve. This space was filled with a homogeneous pinkish fluid. The arachnoid itself showed edematous infiltration. Circumscribed transudation in the perilymphatic spaces around the sacculus could be seen.

Histologic Observations on the Left Petrous Bone: The same findings were noted as in the right, but in addition to vaginal dropsy of the cochlear nerve a similar condition (vaginal dropsy) of the facial nerve was seen (fig. 6). In

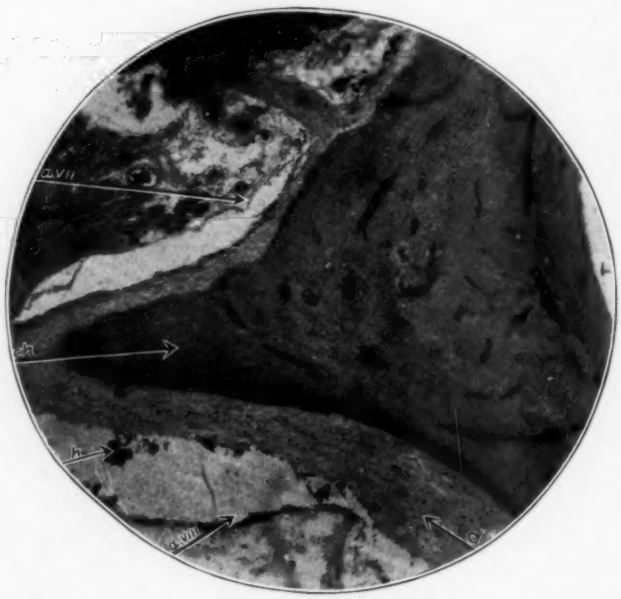

Fig. 6.—Photomicrograph from a case of frontal tumor; section through the internal meatus with the crista horizontalis (*c.h.*) separating the sheath of the cochlear nerve below from the facial nerve above. The arachnoid sheaths (*a.VII*, *a.VIII*) are infiltrated with transudate. Between the thickened dura (*d.*) and the lifted arachnoid of the cochlear nerve recent hemorrhages (*h.*) are seen in the subdural space.

the internal meatus recent hemorrhages were seen in the subdural space (between the lifted arachnoid and the thickened dura), and there was a pinkish transudate in the lymph spaces of the vestibule, as well as an edematous infiltration in the connective tissue of the subarcuate fossa. The saccus endolymphaticus was closed by a subepithelial edema, with an enlargement above the lumen (fig. 7). Disseminated hernias of the brain were noted along the middle fossa.

Comment.—From a clinical point of view this case is interesting. Many symptoms (such as headache, dizziness, disturbance of equi-

librium and ataxic gait) pointed to a neoplasm of the posterior fossa. The otologic findings were helpful in localizing the lesion of the brain. The negative reaction to bilateral calorization, the typical past pointing and falling reaction and the absence of disturbances of coordination ruled out the likelihood of a neoplasm of the posterior fossa. As far as the pathologic picture of the congestive inner ear is concerned, multiple hernias of the brain were seen along the middle fossa, causing in certain areas pressure atrophy of the bone. There was also vaginal dropsy of the cochlear, facial and glossopharyngeal nerves. The enlargement of

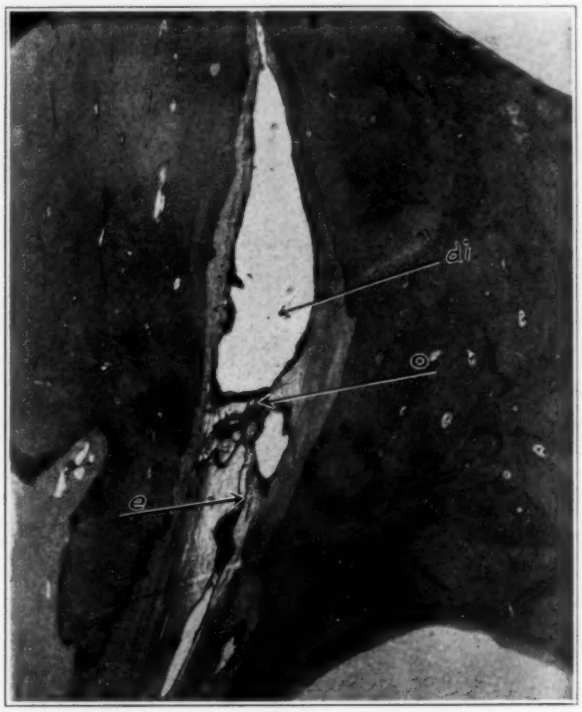

Fig. 7.—Photomicrograph from a case of frontal tumor; section through the saccus and the ductus endolymphaticus. Extreme subepithelial edema (*e.*) in the saccus causes complete occlusion (*o*) of lumen, with resulting distension (*di.*) above.

the aquaeductus cochleae is due to the pressure exerted by the pacchionian granulations. In the membranous inner ear there are beginning organizations, with adhesions and, as a sequela, degenerative changes in the delicate structure of the sense organ.

CASE 4.—A 48 year old white woman entered the hospital presenting headache, malaise, anorexia, dizziness, tinnitus, diminution of the sense of touch and pain on the left side, impairment of the sense of position and movement, astereognosis, hemianopia and choked disks.

Otologic Findings.—The drum membranes appeared normal. Perception of the accentuated whispered voice was 8/20 on the right side and 10/20 on the left. Bone conduction was shortened on both sides. No spontaneous nystagmus was seen, and bilateral calorization yielded negative results, while unilateral calorization showed irritability of both labyrinths. Falling and pointing reactions were typically normal.

Autopsy.—The patient died seven weeks after admission.

Autopsy was reported as revealing glioma of the right parietal lobe invading the deeper structures and extending back into the occipital lobe.

Histologic Observations on the Right Petrous Bone: On the right side were seen engorged blood vessels invading the underlying bones of the pyramid. The

Fig. 8.—Photomicrograph from a case of parietal tumor; section through the aquaeductus cochleae, showing its cerebral ostium (*c.o.*), with ampullar enlargement of the canal. The invading meninges are accompanying the ninth nerve. Meshes and loops are formed by the arachnoid, and two pacchionian granulations (*p.*) are invading the bony wall.

internal meatus showed periosteal thickening. Hemorrhages of the subdural space in the internal meatus and the facial canal were present. The cerebral ostium of the aquaeductus cochleae showed funnel-shaped enlargement. The meninges were evaginated from the posterior fossa into the canal. The dura lined the bony wall; the meshy arachnoid was lifted away from the glossopharyngeal nerve, and between the external sheath of the arachnoid and the epineurium of the nerve

was a space filled with transudate and blood (vaginal dropsy). One could see also two projections, pacchionian granulations, originating from the arachnoid, invading the dura and entering the bony canal (fig. 8). On the tympanic ostium of the aquaeductus cochleae septums of connective tissue were found.

Membranous Inner Ear and Nerve Ganglion Apparatus: Pigmentations in the modiolus and the ductus reuniens were apparent. An increase of perilymphatic tissue in the pars superior of the inner ear was seen. There were adhesions between the sacculus and the medial wall of the vestibule. The ductus endolymphaticus was occluded by connective tissue. Degeneration of the organ of Corti and the nerve ganglion apparatus was evident. The spiral ganglion of the vestibular portion of the cochlea was almost entirely replaced by connective tissue,

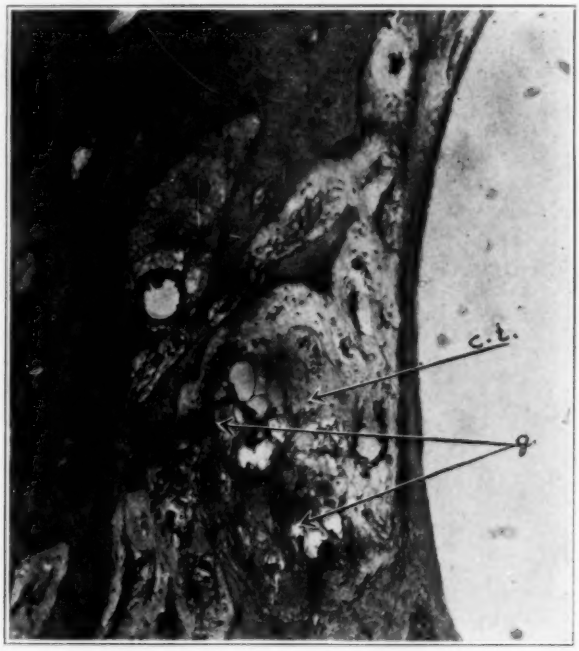

Fig. 9.—Photomicrograph from a case of parietal tumor; section through the spiral ganglion of the vestibular portion of the cochlea, showing degeneration of the majority of ganglion cells (*g.*), with hyperplasia of the interstitial connective tissue (*c.t.*).

so that only a few degenerated ganglion cells were recognized (fig. 9). The spiral ganglion of the middle turn showed only swelling of the interstitial tissue. In the highest turn, the spiral ganglion showed slight changes. Atrophy of the stria vascularis and the vas spirale of the vestibular and the basal turn was present.

Histologic Observations on the Left Petrous Bone: The observations were similar to those on the right side, but there were hemorrhages within the dural layer as well as in the subdural space. The dura of the middle fossa was markedly thickened and contained many endothelial strands and disseminated small hernias of the brain. In various regions of the blood vessel changes of the wall were seen

(atrophy of the media and calcification and hyperplasia of the intima). The cochlear nerve showed disseminated circumscribed degenerations. In the spiral ganglion there was interstitial hyperplasia.

Comment.—In this case advanced changes of congestive inner ear were noted. As a sequel of increased general brain pressure, existing over a long period, secondary processes had occurred, such as alterations in the blood vessels, thickening of the dura of the middle and the posterior fossa with pressure atrophy of the underlying bone and, finally, degeneration of the sense organ and the nerve ganglion apparatus. The latter changes start in the basis of the cochlea (the vestibular and

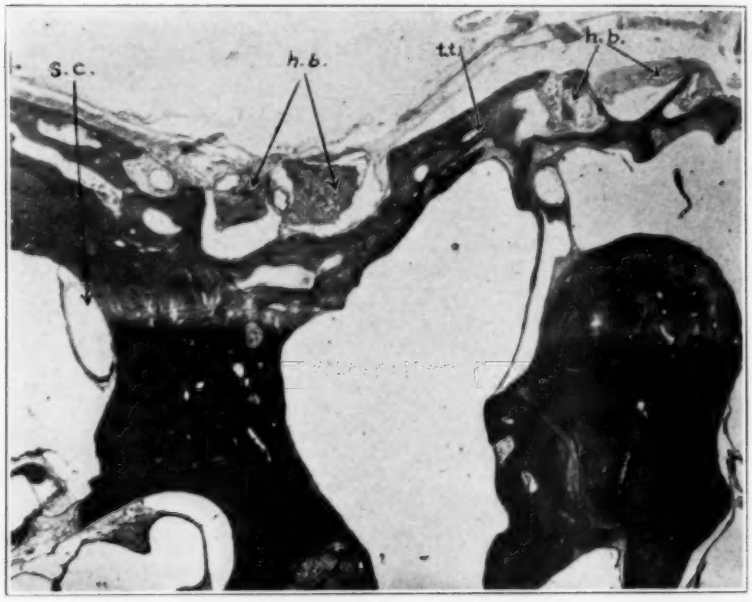

Fig. 10.—Photomicrograph from a case of occipital tumor; vertical section through the middle and inner ear, showing multiple hernias of the brain (*h.b.*) in the middle fossa above the tegmen tympani (*t.t.*) and the semicircular canals (*s.c.*).

the basal portion contain the highest degree of atrophy) and extend to the top in decreasing degrees.

Case 5.—A 45 year old white man entered the hospital with the following symptoms and signs: occipital headache, weakness, dizziness, spots before the eyes, tenderness to percussion over the right side of the occiput, right hemianopia and bilateral choked disks.

The roentgen findings revealed increased endocranial pressure.

Otologic Findings.—The drum membranes appeared normal. Perception of the accentuated whispered voice was 5/20 on both sides. Bone conduction was

shortened, and perception of the upper tones (c-4) was markedly diminished on both sides. There was no spontaneous nystagmus. Calorization showed labyrinthine irritability on both sides. The falling and pointing reactions were typically normal.

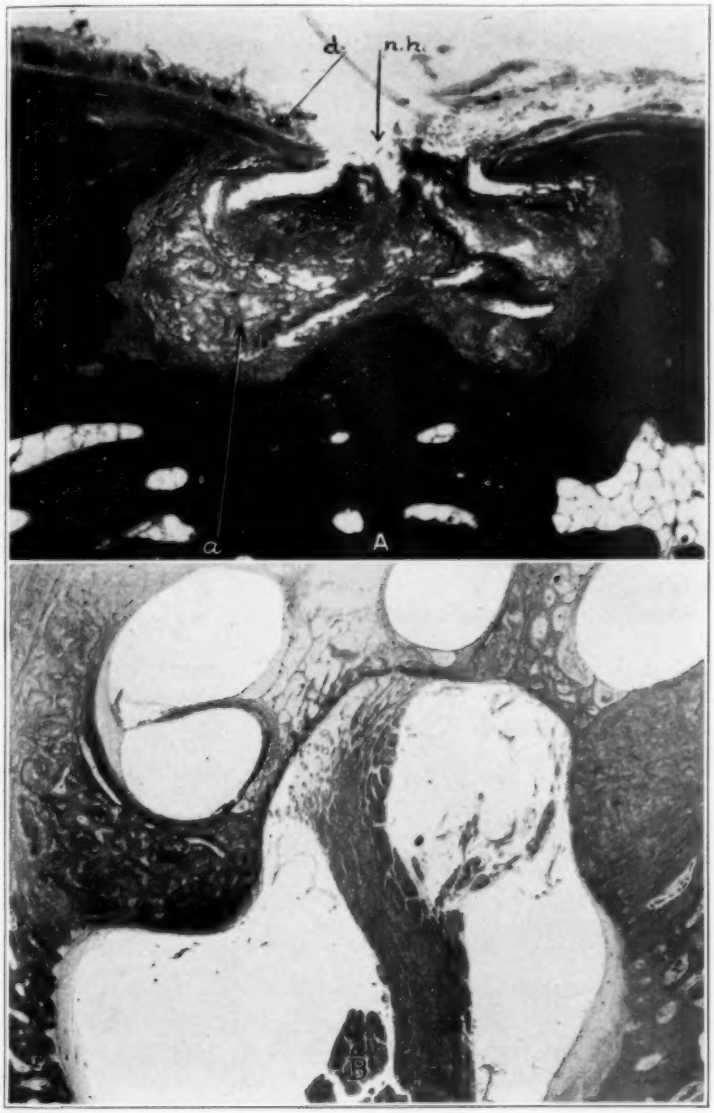

Fig. 11.—*A*, photomicrograph from a case of occipital tumor; section through the petrous bones, showing a large hernia of the brain piercing the dura (*d.*) of the middle fossa. The neck of the hernia is seen at *n.h.* Meshy arachnoid (*a.*) surrounds the hernia, and marked pressure atrophy of the bone is seen. *B*, photomicrograph from a case of occipital tumor; axial section through the cochlea. The tremendous enlargement of the internal auditory meatus may be compared with the size of the cochlear nerve.

Autopsy.—The patient died five weeks after admission.

Autopsy was reported as revealing a large neoplasm of the right side of the occipital lobe.

Histologic Observations on the Right Petrous Bone: There were extravasation of blood and marked congestion of the blood vessels in the modiolus, the septum scalarum and the medullary spaces of the pyramid. Old hemorrhages in the internal meatus, the facial and the carotid canal were present. Pacchionian granulations invading the internal meatus, causing erosions of the bony wall, could be seen. Multiple hernias of the brain appeared on the anterior surface of the pyramid (above the semicircular canals, above the tegmen tympani and above the roof of the external meatus [fig. 10]).

Membranous Inner Ear and Nerve Ganglion Apparatus: Thready exudates were found in the scala tympani of the vestibular turn. Old hemorrhages and connective tissue were seen in the ductus perilymphaticus, near its tympanal ostium. A large amount of pigment was noticed in the modiolus. The saccus endolymphaticus contained blood pigment and connective tissue. The spiral ganglion of the vestibular and the basal turn was partially replaced by connective tissue. There was degeneration of the sense cells in the vestibular and the basal portion, with secondary atrophy of the stria vascularis and the vas spirale.

Histologic Observations on the Left Petrous Bone: Similar observations were noted, with the following additions: Old hemorrhages were seen in the hiatus spurius of the facial canal. A large hernia of the brain appeared above the frontal semicircular canal, showing the invasion of the brain tissue into the underlying bone. The destruction of bone is seen in figure 11 *A* as a butterfly-shaped defect. The internal auditory meatus was markedly enlarged (fig. 11 *B*). The bony destructions were caused by osteoclasts and Volkmann's perforating canals. Thready exudate was found also in the middle turn of the cochlea.

Comment.—In this case the later stages of congestive inner ear were seen. The main changes were as follows: degenerative atrophy of the sense organ and the nerve ganglion apparatus, with atrophy of the stria vascularis and the vas spirale. Also, because of the increased generalized brain pressure there were multiple hernias of the brain (above the semicircular canals, the roof of the middle ear and the eustachian tube) and pressure atrophy of the petrous bone (the middle fossa and the internal meatus).

CONCLUSIONS FROM THE MICROSCOPIC STUDIES

Examination of the recent serial sections of 10 petrous bones, as well as of the earlier set of 10, results in the conclusion that numerous changes in the inner ear (congestive inner ear) are caused by chronic increased brain pressure. These changes can be classified into the following six main groups:

I. Transudation, exudation and lymphatic congestion (dropsy and edematous infiltration) within the membranous inner ear and the nerve ganglion apparatus (vaginal dropsy of the seventh, eighth and ninth nerves).

II. Hyperemia (especially venous), hemorrhages and alterations of the walls of the blood vessels within the inner ear and the petrous bone.

III. Formative changes within the membranous inner ear (ectasia, stenosis and collapse) in the ductus cochlearis, the ductus endolymphaticus, the ductus perilymphaticus and the ductus reuniens.

IV. Formations of hernias of the brain and pacchionian granulations.

V. Secondary (later) changes: pigmentations, attachments and adhesions in the membranous inner ear and ascending atrophy of the nerve ganglion apparatus.

VI. Pressure atrophy of the bone due to osteoclasts and Volkmann's perforating canals, caused by herniations of the brain and pacchionian granulations.

A more detailed discussion of these groups may be desirable.

Group I (Transudation, Exudation, Etc.).—The amount and extent of the fluid vary from small circumscribed perineural and perivascular accumulations within the internal meatus to extensive dropsy within the perilymphatic and endolymphatic spaces of the pars inferior and the pars superior of the inner ear. Furthermore, edematous infiltration of the tissues is seen, as in the ligamentum spirale or the ganglion spirale, or in the form of subepithelial edema of the lumen of the aquaeductus cochleae.

It is of interest to note the vaginal dropsy of the cochlear, facial and glossopharyngeal nerves, as in cases 1, 3 and 4. The arachnoid is edematously infiltrated, lifted away from the epineural sheath; the space between the two (epineural sheath and arachnoid) is filled with a large amount of transudate (fig. 5 *B*).

Group II (Hyperemia, Hemorrhages and Vascular Changes).—The hyperemia is due mostly to passive venous congestion and occurs within the membranous and bony inner ear. It is particularly marked in the vena aquaeductus vestibuli, the vena aquaeductus cochleae and their tributaries. Engorged blood vessels are seen in the axis of the cochlea (vena spiralis modioli), in the endosteal portion of the scala tympani and in the septum scalarum. The same hyperemia is seen also on the floor and the roof of the vestibule. The vessels of the stria vascularis are markedly dilated and engorged, bulging into the lumen of the ductus cochlearis.

Hemorrhages are seen in all degrees and stages, from a small circumscribed extravasation to a massive hemorrhage, as in figure 2, and from recent to old hemorrhages. There are also hemorrhages found within the pacchionian granulations and within the hernias of the brain as in figure 4 *B*.

The walls of the blood vessels are edematously infiltrated, swollen, thickened or homogeneous. In cases in which endocranial pressure has been increased over a long period the arteries show hyperplasia of the intima and pressure atrophy of the media, particularly in the regions where these arteries run along the underlying bone.

Group III (Formative Changes Within the Membranous Inner Ear).—In this group belong such changes as ectasia, stenosis and collapse of the ductus cochlearis.

In case 1 marked ectasia was indicated by the bulging of Reissner's membrane due to the large amount of transudate within the scalae (figs. 1 and 2). A similar condition was found within the ductus reuniens and the ductus endolymphaticus. In figure 7 complete occlusion of the saccus endolymphaticus due to subepithelial edema of its wall can be distinctly seen, causing funnel-shaped ectasia of the lumen above the occlusion.

In contrast to the enlargement of the lumen (ectasia) one finds, on the other hand, narrowing (stenosis) or complete absence (collapse). These changes are brought about by organization of the exudate with resultant adhesions.

Figure 5 *A* gives an example of stenosis of the cochlear duct due to adhesion of Reissner's membrane to Corti's membrane and the stria vascularis.

Group IV (Hernias of the Brain).—The hernias of the brain were found on the anterior surface of the pyramid in the middle fossa (above the semicircular canals, above the canal for the facial and the superficial petrosal nerves and above the roof of the tympanic cavity and the eustachian tube).

The presence of hernias of the brain was known by older authors, such as Cruveilhier.

In 1872 von Recklinghausen [14] observed that in cases of increased endocranial pressure in which an autopsy was performed the removal of the brain was attended with some difficulty, due to adhesions between the temporal lobe and the dura of the middle fossa. After the removal of the brain he found many small disseminated pediculated pieces of brain tissue which remained attached to the dura (hernias of the brain).

Microscopic investigations of hernias of the brain were made by many pathologists.[15]

Benecke [15a] described the development of the hernias on the basis of increased endocranial pressure causing the rupture of the pia with prolapse of the brain tissue into pacchionian granulations.

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14. von Recklinghausen, F. D.: Verhandl. d. phys.-med. Gesellsch. in Würzburg **5**:9, 1872.

15. (*a*) Benecke, J.: Virchows Arch. f. path. Anat. **119**:60, 1890. (*b*) Blasius, N.: ibid. **165**:504, 1901. (*c*) Wojno, O.: Frankfurt. Ztschr. f. Path. **9**:279, 1912. (*d*) Prym, P.: Deutsche med. Wchnschr. **49**:1145, 1923. (*e*) Franz, I.: Frankfurt. Ztschr. f. Path. **33**:72, 1926. (*f*) Erdheim, J.: Jahrb. f. Psychiat. u. Neurol. **39**:322, 1919. (*g*) Mayer, O.: Ztschr. f. Ohrenh. **75**:95, 1917. (*h*) Brunner.[11a] (*i*) Mehler, L., and Satz, L.: Ztschr. f. d. ges. Neurol. u. Psychiat. **151**:441, 1934.

Erdheim [15f] stated that hernias of the brain are caused by increased endocranial pressure which forces the brain tissue as globule-shaped masses into the pacchionian granulations. In the first stage the hernias do not occupy the granulations entirely, but as the pressure increases the pacchionian granulations become filled entirely, and the arachnoid sheath becomes adherent to the dura.

The hernias of the brain are not found throughout but are localized only where there has been previous development of pacchionian granulations.

Group V (Secondary [Later] Changes).—Scattered pigmentations were found in the scalae, the modiolus, the ductus reuniens, the perilymphatic duct and the floor of the vestibulum.

Adhesions resulting from organization processes were seen. In figure 3 *B* there are adhesions between the inferior surface of Corti's membrane and the inner pillar of the organ of Corti. Other adhesions are seen in figure 5 *A*, between the stria vascularis and Reissner's membrane, extending from the upper angle of insertion to the vas prominens.

The secondary atrophic changes in the sense organ and the nerve ganglion apparatus show extending degeneration. This is particularly shown in cases 4 and 5, in which one sees the most severe degenerative changes in the vestibular and basal turn of the cochlea, less involvement in the middle turn and least involvement or no changes of the cell structure in the top turn. The earliest degenerative changes in the organ of Corti are seen as a loss of the cilia; further degenerations occur with destruction or loss of the ciliated cells but no involvement of the supporting structure. The most severe conditions, then, are atrophy of the stria vascularis and the vas spirale. Within the spiral ganglion there is in the earlier stages only a swelling of the ganglion cells; later degenerative changes are shown as destruction of the cells with increase of the interstitial tissue (fig. 9), and in the most severe form of degenerations almost complete loss of the ganglion cells is noted, with replacement by connective tissue. In the nerve there are various stages of degeneration, extending from involvement of the myelin sheath to replacement by connective tissue.

Group VI (Pressure Atrophy of the Bone).—The bony changes are destructive processes due to increased brain pressure. The bone becomes destroyed by osteoclasts and Volkmann's perforating canals. Circumscribed bony erosions are seen in the regions where pacchionian granulations and hernias of the brain invade the underlying bone (figs. 4 *A* and *B*, 8, 10 and 11 *A*). With generalized brain pressure extending over a long period, diffuse pressure atrophy of the bone is seen. In figure 8 one can notice a funnel-shaped enlargement of the bone of the aquaeductus cochleae near its cerebral ostium, and in figure 11 *B* there is marked enlargement of the entire internal auditory canal.

ANALOGY BETWEEN AURAL CHANGES AND CHOKED DISK

After the histopathologic observations have been described and discussed the question arises whether it is justifiable to designate these changes "congestive inner ear." In other words, can it be stated that there is an analogy between the observations with choked disk and those in the cases described? In order to answer this question one must review the anatomic relation of *the eye* to the cranial cavity (choked disk) and, on the other hand, the relation of *the ear* to the cranial cavity (congestive inner ear).

Anatomic Relation of the Eye to the Cranial Cavity (Choked Disk).—All the theories as to the development of choked disk can be condensed and classified into the following three conceptions:

1. The mechanical theory.
2. The inflammatory theory.
3. The neurotrophic theory.

Most authors believe in the mechanical theory, but they give different explanations of the manner in which the pressure acts.

Von Graefe [16] stated the belief that the sinus cavernosus is compressed, with resultant stasis of the veins, causing papilledema.

A more favored theory is that of Schmitz-Manz, based on the anatomic work of Schwalbe.[17] The latter found a communication between the subarachnoid spaces of the brain and the perichoroidal spaces of the eye by means of the subvaginal space (surrounding the optic nerve). Schmitz-Manz stated the belief that with increased endocranial pressure the subarachnoid fluid is forced into the subvaginal space, resulting in an edema of the nerve head and later causing incarceration and strangulation of that portion. He stated also that if there is any impediment between the two spaces in the foramen opticum (hypophysial tumor) choked disk will not develop even with a high degree of increased brain pressure.

Another mechanical theory was presented by Knies, Behr,[18] Wilbrand [19] and Sänger [20] who stated that the flow of fluid, which physiologically is centripetal (from eye to brain) is slowed and later blocked by increasing brain pressure, causing papilledema and bulging of the

16. von Graefe, A.: Arch. f. Ophth. (pt. 2) **7**:58, 1860.

17. Schwalbe, G.: Lehrbuch der Anatomie der Sinnesorgane, Erlangen, E. Besold, 1885.

18. Behr, C.: Klin. Wchnschr. **7**:1818, 1928.

19. Wilbrand, H., and Sänger, A.: Die Erkrankungen der Papilla nervi optici, in Die Neurologie des Auges, ein Handbuch für Nerven- und Augenärzte, Wiesbaden, J. F. Bergmann, 1912, vol. 4, pt. 2, p. 767.

20. Sänger, A.: Neurol. Centralbl. **24**:98, 1905.

subvaginal space. This theory was supported by Behr, who found a circular strangulation sulcus on the optic nerve.

A purely hypothetic theory is that of Benedict, who stated the belief that the changes are neurotrophic and due to vasomotor disturbances of the sympathetic nerves.

Anatomic Relation of the Ear to the Cranial Cavity (Congestive Inner Ear).—There is still today, after many years, the much discussed question whether there is an open communication between the inner ear and the cranial cavity.

Schwalbe [21] injected methylthionine chloride (methylene blue) under constant pressure into the subarachnoid spaces and found the dye in the perilymphatic spaces of the labyrinth.

Steinbrügge [22] expressed the assumption that there is a communication between the inner ear and the brain by means of the ductus perilymphaticus. The pressure in the endolymphatic space may be less, with resultant depression of Reissner's membrane toward the organ of Corti.

Ostmann [23] stated that the saccus endolymphaticus and the membranous inner ear form a closed intercommunicating system.

Quincke [24] stated the belief that there is direct communication between the subarachnoid spaces and the scala tympani.

Hasse [25] stated that for the flow of perilymph in human adults the internal meatus plays a much greater part than the aquaeductus cochleae.

Rejtoe [26] denied the communication between the cerebrospinal and the perilymphatic fluid. He stated the belief that the perilymphatic fluid is a product of the endolymphatic by osmosis.

Karlefors [27] summarized his great experiments and anatomic studies approximately as follows: Thus formerly (and possibly now) the aquaeductus cochleae had the function of interchange of fluid between the brain spaces and the labyrinth, although in human adults the flow is from the brain spaces into the labyrinth.

Recent workers on this study are T. Kibata,[28] Meurman,[29] Karbowski [30] and Jampolsky.[31] The last stated the belief that the perilymphatic

21. Schwalbe G.: Centralbl. f. med. Wissensch. **7**:465, 1869.
22. Steinbrügge, H.: Arch. f. Ohrenh. **29**:87, 1890.
23. Ostmann, P.: Arch. f. Ohrenh. **24**:35, 1887.
24. Quincke, H.: Arch. f. Anat., Physiol. u. wissensch. Med., 1872, p. 153.
25. Hasse, C.: Anatomische Studien, Leipzig, Wilhelm Engelmann, 1873.
26. Rejtoe, A.: Monatschr. f. Ohrenh. **55**:324, 1921.
27. Karlefors, J.: Die Hirnhauträume des Kleinhirns, Stockholm, P. A. Norstedt & Söner, 1924.
28. Kibata, T.: Ztschr. f. Oto-Rhino-Laryng. (Tokyo), 1927, vol. 33.
29. Meurman, Y.: Acta Soc. med. fenn. duodecim **13**:1, 1930.
30. Karbowski, B.: Monatschr. f. Ohrenh. **64**:687, 1930.
31. Jampolsky, L. N.: Monatschr. f. Ohrenh. **69**:23, 1935.

space can be called a system of sinuses of the subarachnoid space, functioning as outlets for drainage.

Reasons for Accepting the Analogy.—Now having presented the anatomic basis, as well as experimental work, I can turn to the arguments of the authors who deny for anatomic reasons the analogy with the choked disk.

In contrast to these authors, I am of the opinion that in certain respects the anatomic relations are more favorable to congestive inner ear. The eighth nerve does not completely fill the internal auditory meatus in the manner in which the optic nerve does its canal; there are numerous perineural and perivascular lymph spaces, which are especially large in the fundus of the internal meatus and in the region of the root of the lamina spiralis ossea. The wall separating the spaces of the scalae is thin and is formed in part by partitions of connective tissue, so that here also one must take into consideration a communication between the cerebrospinal fluid and the perilymphatic fluid of the inner ear. There exists, furthermore, a connection between the ligamentum spirale and the lymph spaces of the modiolus by means of perivascular channels. Therefore, the following pathways communicate between the cerebrospinal fluid and the spaces of the inner ear: the aquaeductus cochleae, the internal auditory meatus, the perineural and the perivascular spaces, the lymph channels of the bony labyrinthine capsule, the spiral ligament and the pacchionian granulations. In my histologic preparations I was actually able to demonstrate congestive changes following these anatomic paths.

Congestive changes in the inner ear can also be readily explained in terms of the blood supply. The internal auditory artery, which is a branch of the basilar artery, forming the circle of Willis, runs to a great extent without branches and then divides into end arteries. Therefore, a venous stasis and compression of the blood vessels of the inner ear are easily possible. The microscopic slides show such changes as engorgement, venous stasis, thrombi and alterations of the walls of the vessels.

Finally, it is known that every disturbance in the circulation of blood in the inner ear leads to changes in pressure, since the capillaries themselves not only serve for nourishment of the tissue but also secrete the endolymphatic fluid.

This secretion occurs in the stria vascularis of the cochlea, so that even the slightest disturbance in the vessels must soon be noticeable in the inner ear. The process is all the more rapid since the membranous inner ear is enclosed in a rigid bone capsule so that changes in pressure could only be compensated by changes in the oval or round windows.

The objection has been brought that clinically choked disks are seen much more frequently than congestive inner ear. This, however, is readily explained. In many cases of generalized endocranial brain pres-

sure choked disk is not seen; furthermore, a small tumor of the brain can often, particularly when it leads to early hydrocephalus internus, produce marked choked disk, while often a tremendous, extensive neoplasm of the brain will be accompanied by slight papillary edema or none at all. It is known also that choked disk can be present without any complaints. This not only holds for the eye, but can be applied to the ear. Instructive in this connection is my case 1 in which, other than tinnitus, a sensation of fulness, slightly shortened bone conduction, no clinical symptoms were seen; while from the standpoint of pathologic anatomy a series of changes had occurred, such as transudation in the cochlear duct, dropsy, edematous infiltration and perineural and perivascular congestion of lymph. The lack of corresponding clinical symptoms can be explained by the fact that this congestive process had not as yet damaged the delicate sense epithelium or the nerve ganglion apparatus, although the entire organ of Corti was embedded in transudate. However, no one would deny the presence of anatomic changes in this case, despite the lack of marked clinical symptoms.

Finally it can be added that in an otologic examination one is dependent on the subjective answers of the patient as far as concerns perception of the voice, the tuning forks and the audiometer. In examining the eye, on the other hand, the examiner sees the choked disk objectively.

Now that it has been seen that congestive changes of the inner ear accompany generalized endocranial pressure, it is up to the clinician to discover this condition as early as possible. Furthermore, the investigations show that with long-standing generalized brain pressure damage of the sensitive end organ may occur, leading to ascending degenerative processes.

Drawing the clinical conclusions from these anatomic studies, one must bend every effort not only to making the diagnosis early but to applying the necessary operative procedure (extirpation of the tumor or decompression). The later such an operation is performed, the more unfavorable is the prognosis functionally. Once a secondary degenerative process takes place in the sense organ, restitution of function is not to be expected.

SUMMARY

I. The histopathologic changes in the inner ear in cases of increased generalized brain pressure (as seen in serial sections of 20 petrous bones) can be classified in the following six main groups:

1. Transudation, exudation and lymph congestion (dropsy and edematous infiltration) in the region of the membranous inner ear and the nerve ganglion apparatus (vaginal dropsy of the seventh, eighth and ninth nerves).

2. Hyperemia, hemorrhage, changes in the blood vessels of the membranous inner ear and the petrous bone.

3. Formative changes (ectasia, stenosis and collapse) of the ductus cochlearis, the ductus reuniens and the saccus and ductus endolymphaticus and perilymphaticus.

4. Multiple hernias of the brain, especially on the anterior surface of the pyramids, and in the middle fossa, accompanied by pressure erosion of the underlying bone.

5. Secondary (late) changes: pigmentation, adhesions and degenerative atrophic processes of the sense organ and the nerve ganglion apparatus.

6. Destructive osseous changes (pressure atrophy).

II. The pathways by which the brain pressure is transmitted into the inner ear are the subarachnoid spaces of the aquaeductus cochleae, the nerve sheaths in the internal auditory meatus, the pacchionian granulations, the ligamentum spirale, the lymph channels of the bony labyrinthine capsule and the blood vessels.

III. One is justified in drawing an analogy between choked disk and congestive inner ear.

IV. Increased generalized brain pressure may exist over a certain period without damaging the delicate sense epithelium and the nerve ganglion apparatus, even when extensive transudation or exudation is present.

V. Increased generalized brain pressure existing over a long period produces secondary degenerative processes extending from the basis of the cochlea to the top.

VI. It is the duty of the otoneurologist by exact and periodically repeated examinations to discover the earliest stages of congestive inner ear and to give the indications for surgical intervention before irreparable changes occur in the delicate sense apparatus.

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TOMOGRAPHY AND CANCER OF THE LARYNX

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MONTEVIDEO, URUGUAY

Since 1922, Dr. Henri Coutard has worked in Europe, and Drs. Chevalier and Chevalier L. Jackson, Dr. W. Edward Chamberlain and others in America, on lateral roentgenography, which is helpful in the diagnosis of laryngeal cancer and in giving laryngologists a more precise idea of neoplastic growth and its extension.

But even with the valuable help of lateral roentgenography, laryngologists did not have the means for a complete study; they had only two points of view, laryngoscopic and lateral.

A frontal view, or anteroposterior projection, cannot be obtained by the ordinary roentgenographic technic, because the interposition of the vertebrae prevents one from seeing the anatomic details of the larynx, composed, as it is, of tissues of poor roentgenologic contrast.

With tomography, the new technic, by which roentgenograms can be made selective of different levels or sections of the body, I have been able to obtain a frontal view, or anteroposterior projection, of the larynx, without any interfering shadows of the vertebrae; the epiglottis, false cords, ventricles, true cords and trachea are faithfully presented, as in an anatomic specimen made by the pathologist.

In 1936 I [1] published the first anteroposterior tomograph of the larynx, taken with my original equipment, and called attention to the importance of this new method in the difficult study of cancer of the larynx.

Since then, I have given the subject special attention and presented reports to the Société de Radiologie, Paris, in April 1937 and to the Society of Otorhinolaryngology of the River Platte in January 1937.

Because of the special structure of the larynx, composed of symmetric halves, it is easy to appreciate the importance of an anteroposterior projection, which affords a comparative study of both hemilarynxes on the same film, showing by comparison the difference between the normal and the pathologic details of each side.

This frontal view free of vertebral shadows can be obtained by a simple method in which the patient is not troubled by any preparation or manipulation.

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1. Leborgne, F. E.: Anl. d. ateneo de clín. quir., June 1936.

But I must say that, important as tomography may be, it does not supplant the lateral roentgenogram or the laryngoscopic view; the three examinations are necessary to make a complete study of the organ, and the three views are especially important in making an accurate diagnosis on which to determine the therapeutic indications.

The progress of surgery and radiotherapy, and the close competition of these two methods has put before the laryngologist a new problem,

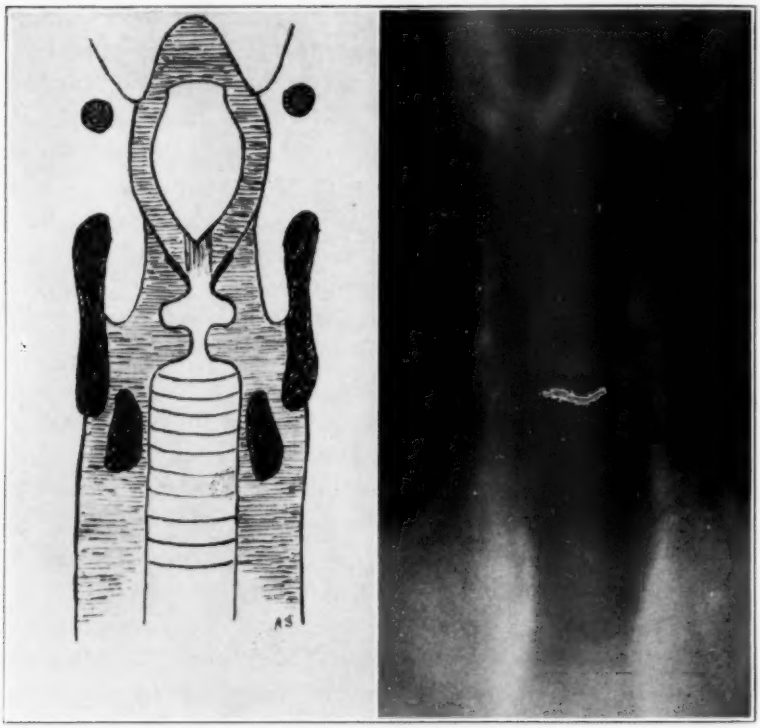

Fig. 1.—Frontal tomograph of a normal larynx, showing the region above the free border of the epiglottis, with the lateral pharyngoepiglottic ligaments, the arytenoepiglottic folds, the laryngeal vestibule and ventricular bands, the ventricles of Morgagni, the two vocal cords, the subglottic region and the tracheal lumen. The negative shadows of the glottic and supraglottic laryngeal lumens together have somewhat the form of a three-leaved clover. The piriform sinuses and the laryngopharyngeal grooves can be well seen. The cartilages appear as if in cross section. The trachea and conus elasticus appear like a candle, the wick of which corresponds to the glottic opening. It should be noted that the printed reproductions are far inferior to the original negative films.

namely, the choice of the best treatment in each particular case. Only a perfect knowledge of the location and extent of the neoplastic invasion will enable him to determine before the operation which of the

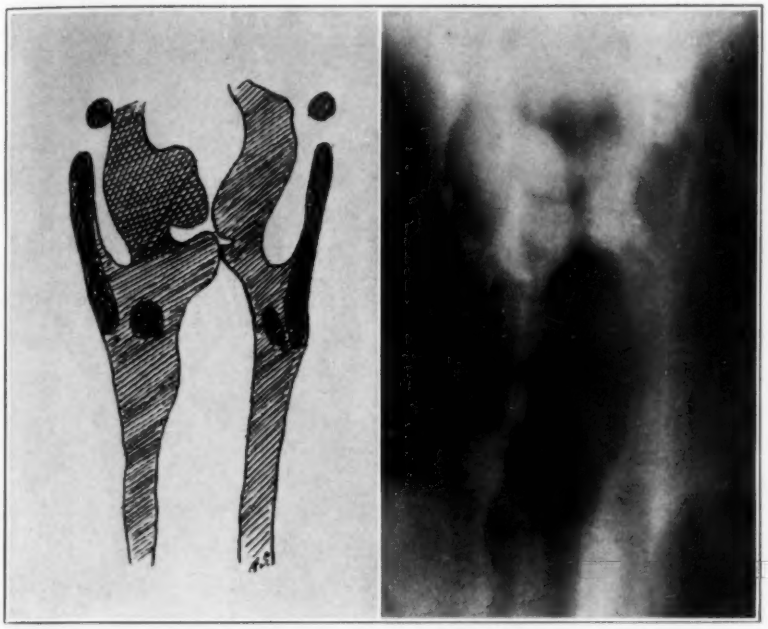

Fig. 2.—Enlarged right ventricular band projecting into the piriform sinus of the same side. The right ventricle is almost obliterated (carcinoma); the right cord is enlarged. The left ventricle is almost obliterated (edema). The patient was operated on by Professor Alonso, and all the roentgen findings were thus confirmed.

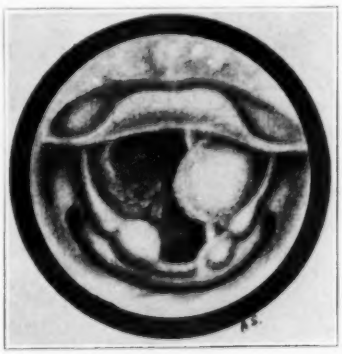

Fig. 3.—Epithelioma of the right ventricular band as seen in the mirror. Ulcerated infiltration of the band is visible, with a zone of necrosis next to the epiglottis; the left band is markedly edematous. (The patient was referred by Dr. Apolo.)

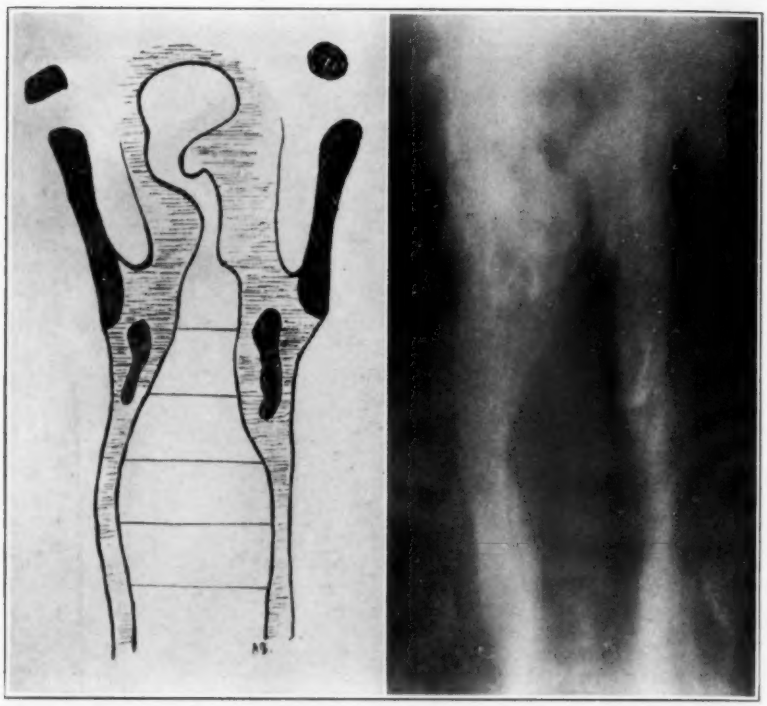

Fig. 4.—Frontal tomograph of the larynx shown in figure 3. The deformity of the laryngeal lumen corresponds with the laryngoscopic image. The ventricles are obliterated.

Fig. 5.—Epithelioma of the left ventricular band, mirror view. (The patient was referred by Dr. Elías Regules.) Slightly productive infiltration was limited to the band, with beginning invasion of the epiglottis. Laryngeal motility was impaired on the left side.

different types of laryngectomy is preferable or whether radiotherapy is advisable. In deciding whether to advise radiotherapy he should give due consideration to the psychic and social effects of the mutilation by surgical treatment of such an important organ as the larynx.

After studying a normal tomographic film, it is not difficult to understand how the neoplasms of the larynx are so clearly visible on the tomographic pictures. Every one knows that lateral roentgenograms clearly show hypopharyngeal tumors and that it is difficult for several reasons thus to study the false cords, ventricles and glottic and sub-

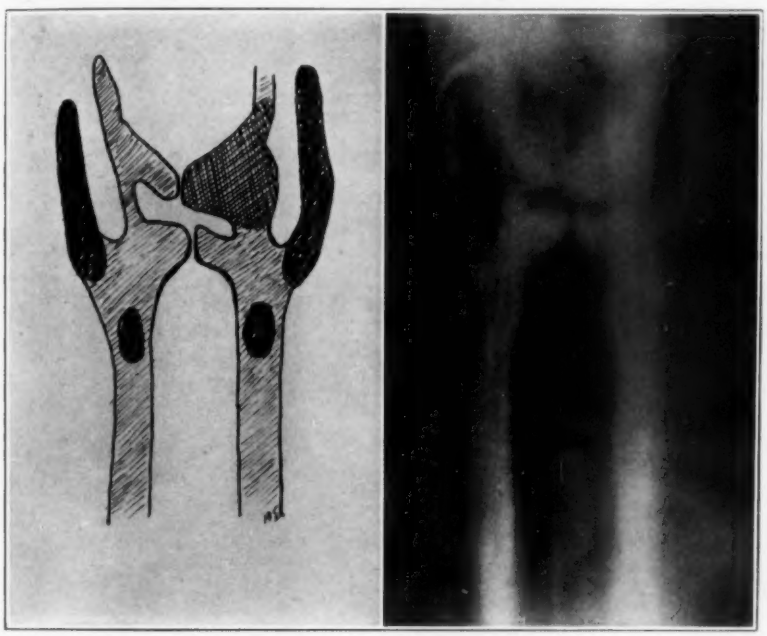

Fig. 6.—Frontal tomograph of the larynx shown in figure 5. The tumor of the band is evident, deforming the laryngeal lumen and filling the corresponding ventricle. There is evidence also of beginning invasion of the left piriform sinus that could not be seen with the laryngeal mirror.

glottic regions; this is where tomography renders the most valuable service.

LARYNGOCELE, OR HERNIA OF THE VENTRICLE OF MORGAGNI

Tomography has enabled me to study in a special way a rare disease of the larynx: laryngocele, or hernia of the ventricle of Morgagni. This is extremely important, as this condition can now be differentiated from laryngeal tumor, for which it has generally been mistaken, especially when the herniated tissue has not extended to the soft parts of the neck.

In 3 of the 5 cases I have seen, the condition was not associated with tumor; in 1 it accompanied a tumor of the subglottic region, and in the other, a tumor of the false cord.

The accompanying illustrations show how accurately the diagnosis of laryngocele can be made and how easily the anomaly can be defined. A patient of Dr. Henri Coutard, of the Chicago Tumor Institute, had been treated by roentgenotherapy for a subglottic tumor, which disappeared after five months although a tumor of the false cord remained; ulceration was not visible, and motility was unimpaired. Examination was made with my tomographic equipment, and I obtained interesting tomographs, which were of real diagnostic value.

Figure 7

Figure 8

Fig. 7.—Frontal tomograph of the larynx showing a large tumor of the right side. Note the encroachment on the right piriform sinus.

Fig. 8.—Frontal tomograph showing a laryngocele of the right side of the larynx. Tomography is of great value in diagnosing this condition.

TECHNIC

The interpretation of laryngeal tomographs is not difficult, but it is absolutely necessary to have perfect tomographic films, and one has to take several films at different levels, in accordance with certain fundamental principles.

In the first place, the laryngeal cuts must be accurately parallel to the axis of the larynx and trachea, particularly in the study of the subglottic region, because diagonal cuts bring forth apparent alterations of the laryngotracheal lumen due to defective technic. The cuts must be frontal so as to afford a comparative examination of both hemilarynxes on the same film.

These are difficult results to obtain, because the trachea and larynx are not always straight. For this reason skill and training are necessary in order to obtain laryngeal tomographs that fulfil these requirements. Great precision is necessary. Experience is important also in the correct interpretation of the films and especially in differentiating between pathologic shadows and those due to errors of technic.

There are anatomic features that help in interpretation. The cartilaginous elements, the thyroid wing and especially the cricoid ring show the exact level and distance that separates them from the endolaryngeal mucosa.

Certain tomographs may be used and may be valuable for diagnosis even if the picture of the trachea is not quite correct, provided one can differentiate the distortions due to the geometric causes previously explained.

It is absolutely necessary that the tomographic features be compared with those of the lateral roentgenogram and the laryngoscopic view.

It is my opinion that from such a plan of study one can obtain exact and valuable information on the pathologic conditions in a particular case of cancer of the hypopharynx or larynx.

RADICAL MASTOIDECTOMY

ITS EFFECT ON HEARING

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AND

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The aural surgeon contemplating the performance of a radical mastoidectomy must ask of himself and discuss with his patient the following questions: What dangers are involved in neglecting the chronic suppuration of the ear? What chances are taken of injuring the facial nerve during the operation? What assurance can be given the patient regarding freedom from aural discharge following operation? What change will there be in the hearing? Much information is available in reference to the first three questions. The otologist can determine with considerable accuracy the imminence of dangerous complications from his knowledge of the progression of pathologic changes involved in the chronic suppurative process. He can assure his patient that, barring rare accidents and unforeseen complications, the facial muscles will retain their normal functions after operation. The pathologic changes manifest in the tympanum, the throat, the nose, the paranasal sinuses and the nasopharynx allow him to venture a shrewd prognosis in respect to the cessation of the aural discharge subsequent to operation. Although none of these questions can be answered definitely and with positive assurance, it seems that the least information is available to assist in determining the effect of the operation on hearing.

Keen [1] investigated the end results in 60 cases of radical mastoidectomy with reference to residual hearing as tested by the use of the watch, the whispered and the spoken voice. In his series of cases a relation between the duration of the chronic suppuration of the middle ear and the postoperative hearing was not apparent. He complained that many recorded preoperative hearing tests were valueless because of the great variation in results produced by a small amount of discharge in the middle ear. It was found that patients presenting gross evidence of cholesteatoma in the middle ear had the poorest preoperative hearing, and the least residual postoperative hearing was in persons

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1. Keen, J. A.: An Investigation of the End Results of Sixty Cases of Radical Mastoid Operations with Special Reference to Hearing, J. Laryng. & Otol. **41**:145 (March) 1926.

with decreased bone conduction. Keen concluded that the main factor influencing postoperative hearing following radical mastoidectomy was the type and amount of tissue covering the promontory. The best postoperative hearing was obtained for patients with a thin, reddish, dry lining of the radical mastoidectomy cavity. The poorest hearing was found in those with an open eustachian tube and a moist thick lining of mucous membrane in the middle ear.

White,[2] in reporting on 100 radical mastoidectomies, stated that 57 of the cavities were dry and well epidermized, 22 were moist and 21 had granulation tissue and a profuse discharge. He determined that his best results were obtained for persons with chronic suppurative otitis media of five to fifteen years' duration. Hearing tests with the whispered and the spoken voice revealed improved hearing in 37, unchanged hearing in 14 and postoperative loss of hearing in 49

Pressman [3] stated that the prognosis for hearing following radical mastoidectomy depends on the state of the auditory nerve, the mobility of the stapes and the opportunity for postoperative care. He reiterated the statement that the better postoperative hearing is to be found in ears with good bone conduction.

Fowler,[4] in presenting 54 cases of radical mastoidectomy in which preoperative and postoperative audiograms were made, maintained that the average radical mastoidectomy does not benefit the hearing. In his series of studies there was an average loss of hearing of 5 decibels in the conversational range. He contended that the modified radical mastoidectomy is of little value in increasing the hearing after long-standing chronic suppuration of the middle ear, because the damage that has been done to the conduction mechanism by the time operation is required is permanent. He made a plea for the avoidance of radical mastoidectomy by early and adequate drainage of acute suppurative processes in the mastoid.

In an effort to gain further knowledge of the effect on hearing of radical mastoidectomy, 50 cases in which the operation has been performed have been analyzed. The tabulated data include the age and sex of the patient, the ear involved, the duration of the otorrhea, the condition of the static labyrinth as determined by caloric stimulation, the preoperative hearing as tested by the whispered and the spoken voice and by the 2-A audiometer and the residual hearing following radical mastoidectomy as tested by the same means. The time after operation that the final tests were made varied from six months to

2. White, L. E., Jr.: Radical Operation of the Mastoid, Arch. Otolaryng. **8**:32 (July) 1928.

3. Pressman, J. J.: Residual Hearing After Radical Mastoidectomy, Laryngoscope **41**:808 (Dec.) 1931.

4. Fowler, E. P.: Hearing Before and After Radical Mastoidectomy, Arch. Otolaryng. **26**:387 (Oct.) 1937.

two years. At the time of the final hearing test 84 per cent of the ears were dry and completely healed while 16 per cent showed some mucous discharge from the eustachian tubes and were labeled tubal ears, although it was too early in some of the cases to abandon hope of obtaining a dry ear eventually. The 50 cases reported are not chosen cases; they are consecutive in the sense that they are those of the 50 persons who were able to return to the clinic for check-up hearing tests of 100 consecutive patients submitted to radical mastoidectomy who were requested to return for them.

From the compiled data, an analysis of the cases was made in order to demonstrate possible significance in the following relations:

I. The duration of otorrhea to the preoperative loss of hearing.

II. The age of the patient to the postoperative residual hearing.

III. The duration of otorrhea to the postoperative residual hearing.

IV. The preoperative labyrinthine response to the postoperative residual hearing.

V. The time required for complete healing of the cavity to the postoperative residual hearing.

VI. The preoperative loss of hearing to the postoperative residual hearing.

In the following paragraphs the loss of hearing is the average loss in decibels of hearing for the tones 256, 512, 1024 and 2048 double vibrations per second, the tonal range generally utilized in conversation.

I. The duration of the otorrhea seemed to have little effect on the preoperative loss of hearing. In a group of 12 cases in which the duration of the suppuration of the middle ear was between two and six years, the average loss of hearing in the conversational range as determined by means of the 2-A audiometer was 37 decibels. In the 10 cases in which the duration was between six and ten years, the average loss was 44.9 decibels. The average loss fell to 37.6 decibels in the 7 cases in which the duration was between 10 and 15 years and then rose again to 45.6 decibels in the remaining 21 cases, in which it was of still longer standing. The one finding of significance that is noted in these figures is that in the majority of cases of chronic suppurative otitis media and mastoiditis of such a character that a radical mastoidectomy is indicated the average loss of hearing is between 35 and 45 decibels.

II. A study of the postoperative hearing in the different age groups demonstrates no variation of consequence except in patients over 45. Of the patients under 45 (44 cases), 46.4 per cent showed an average gain of 8.1 decibels; 43.8 per cent had an average loss of 7.6 decibels, and 9.8 per cent suffered no change. Of the patients over 45 (6 cases), however, 1 (16.7 per cent) gained 15 decibels, while 5 (83.3 per cent) suffered an average loss of 16 decibels.

III. The duration of the otorrhea seemed to influence the residual hearing only in the cases in which the discharge was of less than four years' standing. In 7 such cases, an average gain of 1.5 decibels appeared in 28.6 per cent; in 14.4 per cent the hearing remained unchanged, and in 57 per cent an average loss of 10.5 decibels was found. In the remaining 43 cases the figures were not significant, an average gain of 9.8 decibels appearing in 44.4 per cent, an average loss of 8.3 decibels in 48.7 per cent and unchanged hearing in 6.9 per cent.

IV. The preoperative labyrinthine response was noted in 45 cases, in 30 of which a normal response to caloric stimulation was given, there being 4 cases of hyperactivity, 5 of hypoactivity and 6 in which a response could not be obtained. Although greater variations in residual hearing were noted in the cases in which abnormal labyrinthine responses were given, the figures are not of statistical value.

V. The time required for complete healing of the cavity seems, from the figures here available, to have some bearing on the postoperative residual hearing. Of 7 cases in which complete healing occurred in less than eight weeks, an average gain of 12 decibels appeared in 57 per cent and an average loss of 8 decibels in 43 per cent. Although this variation is not remarkable, it does show an increase in hearing which is above the average for the group. Of the 5 cases in which more than twenty-five weeks was required for complete healing, an average decrease in hearing of 7.2 decibels following operation was demonstrated in each.

VI. The most striking figures were obtained from a study of the preoperative loss of hearing in relation to the postoperative residual hearing. Of 11 cases in which there was an average loss of hearing in the conversational range of from 18 to 30 decibels, no postoperative change in hearing appeared in 27.3 per cent, while an average loss of 8 decibels appeared in 72.7 per cent. Not until a preoperative loss of hearing of over 40 decibels was observed did the average gain in hearing exceed the average loss. As the preoperative loss of hearing increased, it was noted that the residual hearing showed greater gain. Of the cases (5 in number) in which the preoperative loss of hearing was between 60 and 70 decibels, an average of 15 decibels in hearing was gained after operation in 60 per cent, while an average loss of but 2 decibels appeared in 40 per cent.

SUMMARY

One hundred consecutive patients subjected to radical mastoidectomy were requested to return for check-up hearing tests. Of these 50 have returned to date. An analysis of the cases has been made in an effort to determine what factors, if any, influence changes in the acuity of hearing following operation.

The duration of the otorrhea bore no significant relation to the preoperative loss of hearing in the cases studied.

Patients over 45 in the group studied showed the greatest postoperative loss of hearing.

Residual hearing was found to be considerably diminished in patients in whom the otorrhea had existed less than four years prior to radical mastoidectomy.

Preoperative labyrinthine irritability seemed to bear no relation to postoperative changes in hearing in the cases analyzed.

The figures obtained, although not conclusive, suggested that rapid healing of the wound carries a better prognosis in regard to residual hearing. The cases of extremely slow healing were consistent in the appearance of postoperative loss of hearing.

The greatest postoperative hearing loss was sustained in the cases in which the preoperative hearing was best. Conversely, it was noted that the greatest average postoperative gain in hearing resulted in the cases in which the preoperative loss of hearing was more than 40 decibels in the conversational range.

Of the 50 instances recorded, complete healing with freedom from discharge resulted in 84 per cent; in 16 per cent some mucoid discharge originating in the eustachian tube continued.

In the entire group it was found that in 44 per cent of the cases a postoperative gain in hearing averaging 5.6 decibels appeared, in 8 per cent no change in hearing followed operation and in 48 per cent a postoperative loss of hearing averaging 9.4 decibels was experienced.

CONCLUSIONS

Although an analysis of 50 cases does not allow conclusive statements, it is believed that a study of this type reveals information of some value.

Preoperative loss of hearing seems to be determined to a considerable degree relatively early in the chronic suppurative process.

A relatively poor prognosis for residual hearing after a radical mastoidectomy may have to be given to persons over 45 or 50, to those in whom the infection of the middle ear and mastoid is of short duration and also perhaps to those who have comparatively good preoperative hearing as indicated by an average loss in the critical frequencies of less than 25 or 30 decibels.

In the consideration of radical mastoidectomy for a patient in whom the indications for the procedure are clear, the possibility of decreased residual hearing should not act as a deterrent. In the majority of cases the chances that the hearing may be slightly improved or slightly impaired are about equal. In either event the average change is less than 10 decibels.

VALUE OF ENCEPHALOGRAPHY IN THE DIAGNOSIS OF OTOGENIC INTRACRANIAL COMPLICATIONS

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Since Symmonds [1] established the syndrome of otitic hydrocephalus, in 1931, a great number of cases have been recorded in the literature which seem to support his conception of an increase of intracranial pressure stimulated by the focus of otitis media in children or adolescents.

The primary symptoms recorded are the increase of intracranial pressure combined with vomiting, papilledema and headaches, while the spinal fluid is always found clear. The majority of patients recover under adequate treatment.

The symptoms which, according to Symmonds, are significant for otitic hydrocephalus are present not only in this entity but also in (1) edema of the brain, (2) swelling of the brain (3) serous meningitis and (4) nonsuppurative encephalitis.[2] All these conditions are occasionally caused by otitis media. A few words are in order to differentiate between edema and swelling of the brain. In swelling of the brain the amount of solid constituents of the cell is increased, the amount of fluid contained remaining unchanged. In edema of the brain the amount of fluid, intracellular and intercellular, is increased as in any other edema. The pathology of the other diseases need not be mentioned except otitic hydrocephalus sui generis.

It has been stated that real otitic hydrocephalus can occur only when latent hydrocephalus is present, which may become apparent through the stimulation of the aural disease, and produce the symptoms attributed to this entity.

It is only of late that the technic of encephalography [3] and ventriculography has been employed to ascertain the clinical diagnosis, although

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1. Symmonds, C. P.: Otitic Hydrocephalus, Brain **54**:55-71, 1931; Otitic Hydrocephalic and Focal Cerebral Symptoms in Relation to Thrombophlebitis of the Dural Sinuses and Cerebral Veins, ibid. **60**:531-550, 1937; Otitic Hydrocephalus: A Report of Three Cases, Brit. M. J. **1**:53-54, 1932.

2. Not all authors recognize the existence of otogenic nonsuppurative encephalitis.

3. Jerlang, E.: Otitic Hydrocephalus, Hospitalstid. **91**:1028-1040, 1938.

Brunner [4] pointed out the necessity of making use of this diagnostic aid some years ago. A number of case reports [5] show that when intradural surgical intervention was performed in the cases described as those of otitic hydrocephalus a gush of fluid was encountered in opening the subdural spaces but no further pathologic condition was found and recovery ensued. Furthermore, in all the reported cases in which encephalographic examination was performed, enlargement of the ventricles was not found, but there was evidence of an increase of the cisternal spaces, so that it seems that otitic hydrocephalus is more external then internal.

Clinically, it is important to decide whether surgical intervention on the brain is indicated in a case of aural disease with evidence of intracranial pressure, in other words, whether one is concerned with an abscess of the brain or with one of the nonpurulent diseases just mentioned. A further differential diagnosis of any of the complications cannot be decisive, as treatment and prognosis in all these conditions are essentially alike. But even in this respect encephalography is frequently helpful. One is aided in coordinating the procedure with all other signs and symptoms and in being enabled to differentiate otitic hydrocephalus from swelling and edema of the brain and, in turn, these conditions from serous meningitis and nonsuppurative encephalitis. In otitic hydrocephalus there is an increase of spinal fluid either in the subarachnoid spaces (external) or in the ventricles (internal hydrocephalus). Swelling and edema of the brain evidence occlusion of the subarachnoid spaces of the diseased side, the ventricle not being markedly changed. Nonsuppurative encephalitis and serous meningitis do not produce marked changes in the amount of spinal fluid, and the encephalogram probably shows normal findings. In contrast, abscess of the brain produces compression of the lateral ventricle.

In the following description, I wish to report a case in which encephalographic examination performed at the proper time would have

4. Brunner, H.: Zur diagnostischen Bedeutung der Enzephalographie bei otogenen Hirnkomplikationen, Wien. klin. Wchnschr. **47**:199-202, 1934; Otogene intrakranielle Komplikationen, in Bumke, O., and Foerster, O.: Handbuch der Neurologie, Berlin, Julius Springer, 1928, vol. 10, pp. 194-267.

5. Ersner, M. S., and Myers, D.: Otitic Hydrocephalus with a Suggestion as to Its Etiology, Ann. Otol., Rhin. & Laryng. **45**:553-566, 1936. Scal, J. C.: Otitic Hydrocephalus (Pseudomeningitis): Report of a Case, Laryngoscope **42**:936-942, 1932. Smith, C. H.: Otitic Hydrocephalus, with Report of a Case, ibid. **44**:931-934, 1934. Garland, H. G., and Seed, G. S.: Otitic Hydrocephalus, Lancet **2**:751-753, 1933. Carr, F. C.: Report of a Case of Acute Mastoiditis Complicated by Internal Hydrocephalus, Laryngoscope **41**:635-637, 1931. MacAlpine, D.: Toxic Hydrocephalus, Brain **60**:180-203, 1937.

prevented intracerebral surgical intervention and in which after the procedure had been carried out some healing influence was evidenced, such as it is known to possess for meningitis and epilepsy.

REPORT OF A CASE

M. R., a Negro, 15 years of age, was operated on for acute mastoiditis with recurrent otitis media of the right ear on March 29, 1939. The lateral sinus was exposed, situated directly in the antrum, and was found covered with granulations. However, his progress was uneventful and he was allowed to leave the hospital. On April 19, he reappeared at the dispensary with complete paresis of the right facial nerve. Radical operation on the right ear was performed and the dura exposed in the antrum. Three days later a temperature of 104 F. and horizontal-rotatory nystagmus to the right developed. Spinal puncture gave normal results. The temperature rose during the next few days to 105 F. and decreased slowly under the administration of neoprontosil. During this time the patient was stuporous but showed no further signs of intracranial involvement until May 1. He then had an epileptiform convulsion followed by severe headaches. Spinal puncture on the following day revealed increased pressure but clear fluid; the Pandy reaction was slightly positive, and there were 10 cells. There was slight hemiparesis on the left and choking of the disks from 1.5 to 2 D. Under the impression that an abscess of the right temporal lobe was present, the brain was exposed, but pus was not encountered. Postoperatively, the patient was irritable, and he returned to his previous stuporous condition soon thereafter. Two days later marked disturbance in the coordination of the left upper extremity and flaccid paresis of the arm and wrist developed. There was coarse nystagmus to the right. All reflexes were normal with the exception of the left triceps and radial, which were absent. In addition, the patient complained of a deep pain in his right eye. The following day encephalographic examination was performed to exclude an abscess of the brain. The temperature was around 102 F. during all this time. Slight paresis of the abducens nerve developed on the right side. Nystagmus and choking of the disks persisted, and on May 7 slight twitching and later central paresis of the left facial nerve was noted. The tongue deviated to the left, and the hemiparesis became more marked. There were no pathologic reflexes. A roentgenogram of the cervical part of the spine revealed the existence of a left cervical rib.

As the clinical course still seemed to point in the direction of an abscess of the brain, another encephalographic examination was performed on May 8. Six hours later the patient had three consecutive epileptic attacks of the jacksonian type, confined to the left side. From then on, his temperature returned to normal; all neurologic symptoms disappeared; the choking of the disks subsided rapidly, and recovery was uneventful. Two months after the third operation, the ear was dry and epithelized; the paresis of the facial nerve on the right side was subsiding under faradic stimulation. In both encephalographic examinations, the fluid was found clear; the pressure was increased but otherwise normal. Thirty cubic centimeters was removed in the first and 35 in the second, each amount being replaced by 5 cc. less air. A third filling was performed before the discharge of the patient, about three weeks later.

It seems advisable to discuss the encephalographic findings as one. In all the films one has to differentiate two points, the filling of the ventricular system and that of the subarachnoid spaces. The first filling showed a marked contrast in

the quantity of air contained in the ventricles in the anterior-posterior and in the posterior-anterior position of the head. The right ventricle was better filled than the left, irregular but not markedly enlarged. The subarachnoid spaces did not evidence filling on the right side but revealed excellent filling on the left. The lateral view showed the left ventricle well filled with the exception of the inferior horn, while the middle cella on the right side was incompletely filled. Similar films resulted from the procedure three days later. Encephalographic examination three weeks later, before the discharge of the patient, showed both ventricles ideally equal and the subarachnoid spaces well filled. The incomplete filling of the middle cella of the diseased side observed in the first two encephalographic examinations suggested the presence of a blocking-off process but was only the result of the use of a small amount of air, as with a change in the position of the head air passed freely through the ventricles, while the entire subarachnoid space of the affected side could not be filled by moving the head.

COMMENT

In my case, a rise of temperature during two weeks, the increase of leukocytes in the blood, the rise of intracranial pressure, headache, papilledema and, finally, signs of left hemiparesis led to a tentative diagnosis of an abscess of the right temporal lobe. Exploration of the middle fossa was performed under this assumption, but no pus was encountered. Two errors must be admitted in this case, although fortunately they did not cause harm to the patient. 1. An encephalographic examination was not performed before operation. 2. The flaccid paresis of the left arm should not have been accounted for by a central lesion. The first mistake was corrected three days after operation, when abscess of the brain was ruled out. The second point was cleared up when, in looking for possible reasons for peripheral brachial paresis, a cervical rib was discovered. The pressure of the rib on the brachial plexus during the convulsion which preceded the paresis may have caused flaccid paresis of the arm.

Although the focal symptoms persisted and even slight central paresis of the facial nerve developed on the left side, a second encephalographic examination yielding the same results and followed by rapid recovery helped to prove the nonexistence of an abscess of the brain. Whether the recovery was entirely due to the second encephalographic examination is difficult to decide. The favorable turn may have been due to the opening of the blocked arachnoid villi, the drainage of which was interfered with.[6] It is, however, more likely that the irritation of the meningeal vessels and of the brain tissue resulted in an increase in the blood supply to the brain and the removal of toxins and poisons.

6. Hassin, G. B.: Hydrocephalus: Studies of Pathology and Pathogenesis with Remarks on the Cerebrospinal Fluid, Arch. Neurol. & Psychiat. **26**:1164-1184 (Dec.) 1930.

No doubt the decrease of intracranial pressure through spinal puncture and exposure of the dura also had some favorable healing effect.

In the diagnosis in my case I can exclude otitic hydrocephalus with some certainty because of the lack of roentgen findings. Nonsuppurative encephalitis does not cause occlusion of the subarachnoid spaces, which occurred in my case. Serous meningitis is evidenced through changes in the spinal fluid. So either swelling or edema of the brain was present, most probably the latter.

Without doubting the existence of true otitic hydrocephalus I should like to suggest that the term should be restricted to that condition proper, which should be differentiated clinically from other entities producing the same symptoms, so as to avoid the great confusion of names to which Williams[7] has recently called attention, all covering the same clinical picture. As encephalographic examination can be performed in cases of increased intracranial pressure of the middle fossa—with which cases alone this discussion is concerned—without harm to the patient, especially if only a small amount of fluid is removed and air is substituted, I believe it is necessary that it be performed routinely in all cases in which a suspicion of cerebral complications within the middle fossa is raised. The diagnostic help in excluding abscess of the brain and differentiating between nonsuppurative conditions is great. For the clinician, of course, the exclusion of abscess of the brain, which in turn excludes the necessity of intracerebral operation, is sufficient. The treatment of all other conditions is essentially alike, viz., clearing up of conditions of the ear and exposure of the dura of the middle fossa more extensively because of the papilledema. The prognosis with such conditions is generally favorable, and the papilledema usually subsides, even if a higher degree is present. This point is substantiated by a case observed at the Vienna Policlinic.[8] This was the case of a girl of 8 years, in which mastoiditis following scarlet fever was accompanied by papilledema of 6 D. The dura of the middle fossa was exposed more widely, and the papilledema subsided to 3.5 D. four days later and disappeared completely within six weeks. The tentative diagnosis in the case at the time was scarlatinal encephalitis accompanied by mastoiditis.

SUMMARY

In juvenile or adolescent patients suppurative otitis media, papilledema, headache, vomiting and increased pressure of the spinal fluid can be explained, as described by Symmonds and others, as a result

7. Williams, H. L.: The Underlying Factors Concerned in Otitic Hydrocephalus, Tr. Am. Laryng., Rhin. & Otol. Soc. **44**:36-83, 1938.

8. Grabscheid, E.: Scharlachencephalitis, Mastoiditis Concomitans, Monatschr. f. Ohrenh. **66**:1133, 1932.

of otitic hydrocephalus but also as a consequence of (1) swelling of the brain, (2) edema of the brain, (3) serous meningitis and (4) nonsuppurative encephalitis.

Encephalographic examination should be used routinely to differentiate these conditions from abscess of the brain and also to help in distinguishing between them. As a favorable prognosis results from this procedure, the ventricles can also be filled with air in cases of increased intracranial pressure without harm to the patient, if the necessary precautions are observed and the disease is localized within the middle fossa.

A case is reported in which encephalographic examination if performed at the proper time would have prevented surgical cerebral exploration but when performed later in the course of the disease aided in the making and confirming of the diagnosis and in the improvement of the patient's condition, which was most likely produced by an edema of the brain.

The presence of a cervical rib complicated the clinical picture, producing peripheral brachial paresis. Complete recovery ensued.

The use of the term "otitic hydrocephalus" should be restricted solely to that condition as proved by encephalographic examination.

The prognosis, even with a higher degree of papilledema, is favorable even without much decompression of the brain, as proved in a second case, in which a 6 D. papilledema gave way to normal conditions.

CONGENITAL FISTULA OF THE NECK COMMUNICATING WITH THE MIDDLE EAR

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Congenital fistula of the neck has been a subject of wide and varied interest in medicine both in the United States and abroad for the past century. During this time it not only has held the academic interest of the embryologist and anatomist but has been a matter of practical concern to the otolaryngologist, the general surgeon and the internist. This is evidenced by a literature replete with contributions, many in the form of excellent monographs [1] from the representatives of these specialties. The purpose of this paper is not to give a comprehensive review of the literature but rather to add to it the report of a more or less unusual case, in which a fistula of the neck communicated with the middle ear. We have had the opportunity to study this case clinically and histologically. In this report an attempt is made to indicate the derivation of this fistula and to correlate the many unusual anatomic features presented.

REPORT OF A CASE

A 5 month old girl was referred by Dr. S. Karelitz to the pediatric service of the Mount Sinai Hospital because of an infection of the upper part of the respiratory tract. On the day after admission the temperature abruptly rose to 108 F., and the infant died. The cause of the hyperpyrexia was not determined.

At birth a marked deformity of both ears had been observed. During the first few weeks of life the patient had had marked difficulty in feeding, accompanied by regurgitation of milk through the nose and also occasionally through the ears. After this period the feeding had been normal. Pertinent in the family history was the presence of fistulas of the neck in the father, in the father's aunt and in the father's grandmother.

Physical Examination.—The appearance of the face was fishlike. The eyes seemed small, the nose prominent and the chin receding. There was a suggestive webbing of the toes. The trunk and extremities were well developed and showed no other abnormalities.

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1. Leegaard, F.: Arch. f. Laryng. u. Rhin. **26**:125-234, 1912. Marx, H., in Henke, F., and Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1926, vol. 12, pp. 609-734.

Both auricles were microtic. The pinna was represented by a heavy, almost ropelike band, in which the helix and anthelix seemed fused and in which the tragus, antitragus and lobule could be but barely identified. The auricle was lower than usual—at the level of the angle of the jaw. Two fistulas communicated with each other on each side of the head (fig. 1). One of these was observed to begin as a small opening in the anterosuperior part of the malformed auricle, close to the external auditory meatus, and to course backward and downward, making its exit in the neck immediately behind and at the lower margin of the auricle. At this site there was a slitlike opening about 1 cm. in length. A probe could be passed between the two orifices. The second fistula extended from the latter orifice medially and forward into the deep structures of the neck. A second probe could be passed into this fistulous tract for about 4 cm. The external meatus in its outermost part seemed well developed; in its deeper portion, however, it was so narrowed that the drum could not be visualized.

Histologic Examination in Serial Sections of the Right Temporal Bone.—The external auditory canal was much lower than normal. It was considerably narrowed and tortuous in its course inward. It narrowed to a fine fistulous channel and then widened as it approached the middle ear. It was lined by stratified squamous epithelium, undergoing excessive desquamation. Hair and ceruminous and sebaceous glands beneath the epidermis were clearly demonstrable.

The mastoid was undeveloped. It was comprised of cancellous bone marrow. Pneumatization was not evidenced. The antral cavity was relatively small. It was partially occupied by embryonic mesodermal tissue. The roof of the antrum dipped downward considerably, and the digastric and sternomastoid muscles were inserted relatively high. This resulted in a marked narrowing of the antrum from above downward. There was an acute suppurative process in the antrum with ulceration of the mucosa.

The tympanic cavity was separated from the external auditory meatus by a rudimentary tympanic membrane anteriorly and by a plate of bone posteriorly. The origin of the plate of bone could not be determined. It merged with the tympanic and the squamous portion of the temporal bone. The tympanic membrane was relatively thick. It was lined externally by stratified squamous epithelium and internally by cuboidal epithelium in its posterior portion. Anteriorly, however, in close proximity to the eustachian tube, the lining epithelium reversed itself so that the external layer of the drum was cuboidal and the internal layer was stratified squamous epithelium.

The tympanic cavity also was considerably narrowed from above downward. The floor of this cavity was not comprised in its entirety of bone. The jugular dome, which was relatively small, comprised only the posterior part. Anteriorly the floor was formed by muscle tissue, which, because of its high insertion, encroached on the lumen of the tympanic cavity. The ossicles were malformed. The short process of the malleus was inserted on the roof of the external canal. The head of the malleus impinged on the squama of the temporal bone. The head of the incus was ankylosed to the roof of the antrum. Two plates of bone extending from the inner tympanic wall coursed through the tympanic cavity and attached themselves to the squama. The jugular bulb was relatively small and posteriorly was considerably lower than usual.

There was evidence of an acute suppurative process in the middle ear.

A fistulous tract took its origin in the neck below and somewhat posterior to the external canal. It coursed upward, mediad and anteriorly and terminated in the

Fig. 1.—Photograph of the infant, showing the malformed auricle with a probe in each of the two fistulas. One probe (*A*), inserted in the auricular fistula, enters in front of the helix and makes its exit just behind and below the auricle. This site of exit is the point of entrance of the lateral fistula of the neck. A second probe (*B*), inserted in this fistula leads into the middle ear. The photograph was presented by Dr. Karelitz.

Fig. 2.—Photomicrograph; vertical section through the antrum, showing the lateral fistula of the neck, *F*, close to its external opening. At this site the fistula is bifurcated and Y shaped. The external auditory canal, *EAC*, is considerably contracted and the antral cavity, *A*, narrowed.

ESC represents the external semicircular canal; *FN*, the facial nerve and *PSC*, the posterior semicircular canal.

hypotympanum. About midway in its course it divided into two parts (fig. 2), an anterior one, which ended blindly in the neck, and a posterior one, which widened and terminated, as just stated, in the tympanum (fig. 3). The fistulous tract was lined by stratified squamous epithelium undergoing hornification. Beneath the epidermis were seen serous and mucous glands and lymph nodes. As the fistula approached the tympanum it was partially lined by cuboidal epithelium. At that site, however, the inner layer of the tympanic membrane was comprised chiefly of stratified squamous epithelium.

The facial nerve in the tympanum was much lower than usual. Multiple dehiscences in its bony canal communicated both with the dura and with the tympanic

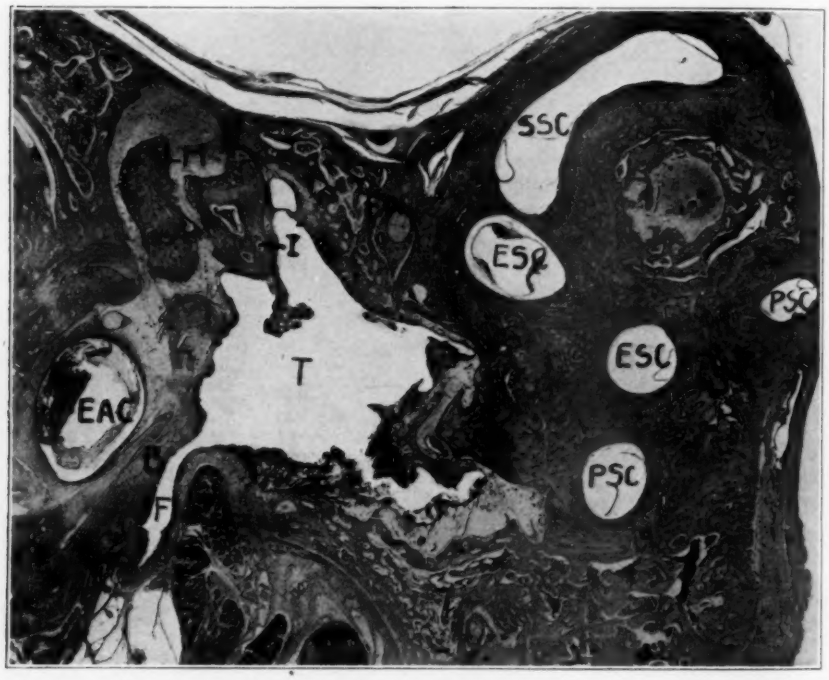

Fig. 3.—Vertical section through the tympanum, illustrating the opening of the fistula into the tympanum. A plate of bone, *B*, ends blindly in the posterior part of the external auditory canal; the malformed ossicles and the formation of new bone may be seen adjacent to the facial nerve.

EAC represents the external auditory canal; *ESC*, the external semicircular canal; *F*, the fistula; *FN*, the facial nerve; *I*, the incus; *M*, the malleus; *PSC*, the posterior semicircular canal; *SSC*, the superior semicircular canal, and *T*, the tympanum.

cavity. The chorda tympani nerve could be identified in the external canal but could not be traced in its course through the tympanum.

The bony and the membranous labyrinth were essentially normal.

COMMENT

The outstanding feature in this case was the presence of a lateral fistula of the neck which communicated with the middle ear. Of further interest was the association of this fistula with a second fistula of the auricle and with congenital malformations of the external and middle ears. These anomalies are no doubt intimately related to the embryonic branchial system.

It is not our intention to give a detailed review of the development of the branchial system. The subject has been adequately covered since the original works of Rathke [2] and Ascherson [3] in 1832. It suffices for the purpose of this case report to recall but a few outstanding features.

The first pharyngeal pouch, represented by the eustachian tube and the middle ear, and the first branchial cleft, represented by the external auditory meatus, are the only portions of the branchial apparatus in man which retain in later life characteristics of their embryonic state. The remainder of the branchial system disappears after the first few weeks of embryonic life, giving rise, however, to important structures which persist throughout postfetal life. Failure of proper development of the branchial system results in malformation of these structures and in the formation of congenital branchiogenetic fistulas. With these preliminary statements we can now discuss in a general way the anomalies observed in this case.

I. *Fistula of the Neck Communicating with the Middle Ear.*—Lateral fistulas of the neck are by no means uncommon. They may appear in members of the same family and through three or four generations. The external opening of these fistulas usually appears along the anterior border of the sternocleidomastoid muscle at any level from that of the mastoid tip to that of the clavicle. The internal opening, when the fistula is complete, communicates with the pharynx and is almost invariably in or near the tonsillar fossa. More often the fistula is incomplete and ends as a blind tract in the deeper structures of the neck. It is generally agreed that the fistulas are derived from the branchial system. There still exists a difference of opinion, however, as to the exact site of their origin. Von Kostanecki and von Mielecki,[4] in a comprehensive review of the subject, ascribed it to the second pharyngeal pouch. On the other hand, Wenglowski,[5] on the basis of his

2. Rathke, cited by Lewis, F. T., and Bremer, J. L.: Textbook of Histology, Philadelphia, P. Blakiston's Son & Co., 1927, p. 223.

3. Ascherson, F. M.: De fistulis colli congenitis adjecta fissurarum branchialium in mammalibus avibusque historia succincta, Berlin, C. H. Jonas, 1832.

4. von Kostanecki, K., and von Mielecki, A.: Virchows Arch. f. path. Anat. **120**:385-436, 1890; **121**:55-87, 1890.

5. Wenglowski, R.: Arch. f. klin. Chir. **100**:789-892, 1912-1913.

painstaking study, stated the belief that they are derived from the embryonic thymic duct, a remnant of the third pharyngeal pouch. Other investigators [6] have implicated other sections of the branchial system. Of especial interest to us, however, are the few reported cases in which the fistulas were considered derivatives of the first branchial cleft. Virchow's often cited case,[7] published in 1865, is a classic example. In this instance the outer opening of the fistula was below and behind a microtic auricle and the inner opening in the nasopharynx, a short distace from the eustachian tube. In König's case [8] also derivation from the first branchial cleft was evidenced; here the external orifice was located at the anterior margin of the sternocleidomastoid muscle about 2 cm. below the auricle. The fistulous tract extended through the substance of the parotid gland and terminated in the external auditory canal.

Communication of a lateral fistula of the neck with the middle ear, observed in the case herein reported, is most unusual. Only 1 similar case, namely, that of Riedel,[9] has to our knowledge been previously reported. In this instance a fistulous tract was resected at operation from the angle of the jaw to the styloid process. Resection of the entire fistula could not be performed, but the belief was stated that on probing 1 to 2 cm. further the middle ear was entered.

A study of the character of the lining epithelium and subepithelial tissue by microscopic examination at times aids in establishing the origin of a fistula. Thus the presence of ectodermal tissue indicates origin from a branchial cleft. On the other hand, the presence of ciliated epithelium only, particularly with underlying lymphoid tissue, suggests that the fistula is derived from a pharyngeal pouch. However, when the fistula is lined by two or more types of epithelium, as in the case described by us, it becomes more difficult to determine its derivation. The presence on histologic examination of stratified squamous epithelium, with its subepidermal structures, lining the fistula in almost its entirety, however, favors origin from a branchial cleft. The close association of the fistula with the malformed parts of the external and the middle ear seems further to localize its origin in the first branchial cleft.

II. *Fistula of the Auricle.*—The communication of a fistula of the auricle with the surface of the neck observed in this case also is most

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6. Carp, L., and Stout, A. P.: Ann. Surg. **87**:186-209, 1938. Hyndman, O. R., and Light, G.: Branchial Apparatus: Its Embryologic Origin and Pathologic Changes to Which It Gives Rise, with Presentation of the Familial Group of Fistulas, Arch. Surg. **19**:410-452 (Sept.) 1929.

7. Virchow, R.: Virchows Arch. f. path. Anat. **32**:518, 1865.

8. König, F.: Arch. f. klin. Chir. **51**:578, 1895.

9. Riedel: Verhandl. d. deutsch. Gesellsch. f. Chir. **26**:125-127, 1897.

unusual. In the vast majority of instances the so-called congenital auricular fistula is in reality a shallow tract which ends blindly.[10] It is the simplest and most common type of developmental abnormality of the external ear. It is believed to be caused by faulty coalescence of the embryonic auricular hillocks first described by His.[11] These hillocks normally arise from the first and second branchial arches and after coalescence assume the form of the auricle.[12] The congenital aural fistula most often is situated at the anterior rim of the helix, is ectodermal and terminates blindly a short distance inward. Rarely is this type of fistula complete. Küttner [13] described an instance in which this condition prevailed. When the fistula is complete its derivation is believed to be from the first branchial cleft rather than from the improper fusion of the auricular tubercles. We are in accord with this view. We are further of the opinion that the auricular fistula in the case reported by us, because of its extent and because of its communication with the second fistula in the neck, can best be explained on the same basis.

III. *Malformations of the Middle and the External Ear.*—The malformations of the external and the middle ear noted in the case under discussion can likewise be attributed to faulty development of the branchial apparatus—the malformations of the external canal, the malleus and incus, the middle ear and antrum and the stapes to faulty development of the first branchial cleft, the first branchial arch, the first pharyngeal pouch and the second branchial arch, respectively.

A review of the congenital anomalies observed in the case described discloses their close embryologic relation, namely, that they are all derived from the first and second divisions of the branchial system.

SUMMARY

A case of congenital lateral fistula of the neck communicating with the middle ear is presented with detailed gross and microscopic studies. Other congenital malformations of the external and middle ear, including a fistula of the auricle, are considered. An attempt to correlate these anomalies and to determine their origin is made.

10. Selkirk, T. K.: Fistula Auris Congenita, Am. J. Dis. Child. **49**:431-447 (Feb.) 1935. Congdon, E. D.; Rowhanouvougse, S., and Veramisara, P.: Am. J. Anat. **51**:439-463, 1932.

11. His, W.: Anatomie menschlichen Embryonen, Leipzig, F. C. W. Vogel, 1885, pt. 3, pp. 211-221.

12. Streeter, G. L.: Contrib. Embryol. (no. 69) **14**:111-128, 1922.

13. Küttner, H.: Deutsche med. Wchnschr. **39**:489, 1913.

IS MEDICAL PHONETICS AN ESSENTIAL PART OF OTORHINOLARYNGOLOGY?

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If an otologist thinks of a speech clinic he has in mind a class of stuttering and lisping children under the care of a speech teacher. In literature he finds articles on stuttering and "nervous speech disorders" written by doctors of philosophy and only rarely one by a doctor of medicine. The university speech clinics are as a rule affiliated with departments of public speaking or dramatics or—if the problem is considered medical—connected with pediatrics and consequently run by teachers. The work done by these departments is excellent—there is no doubt about it.

The great question is: Is phonetics a psychologic problem or a medical one which has been neglected by physicians? Two examples from the otologist's practice shall serve to clarify the situation. After thyroidectomy the recurrent nerve can be impacted or stretched or severed. In the case of an impacted or severed nerve the practitioner certainly can try a new operation—with doubtful success, however. He finds the typical primary symptoms of a paralyzed recurrent nerve: hoarseness, high-pitched voice and waste of expiratory air in speaking. And only because he does not consider phonetics a medical field such patients are frequently neglected. With careful treatment vocal functions could have been restored to a great extent.

If one is familiar with the diagnosis of the different rhinolalias one cannot overlook functional weakness of the soft palate. This manifestation effectuates the same speech symptom as does diphtheria, open rhinolalia. The air escapes through the nose because of the deficient closure of the nasopharynx. Enlarged adenoids act as an absorbing medium partially obstructing the passage of the air through the nose. By removing them carelessly one establishes open rhinolalia. Some weeks later the mothers of such poorly speaking children cannot be put off any longer; the children will never speak normally again.

These are two conditions of the many that the phonetician sees in medical speech clinics. They are medical problems which are frequently overlooked or badly taken care of.

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The equipment of a clinic for diseases of speech and voice proves sufficiently its purely medical basis. It consists of the following articles:

1. The regular outfit of an otorhinolaryngologist. It goes without saying that the basic training of a physician for the speech and voice has to be that of an otorhinolaryngologist.

2. The stroboscop. It makes visible both the vibrations and the level of the single vocal cord.

3. The kymograph. It is used as follows:

(*a*) For the recording of respiration in connection with a thoracic and abdominal rubber belt.

(*b*) For the recording of articulation. The patient speaks into an English narcosis mask connected by a rubber tube with a Marey tambour. A glass olive is fixed in his nose and by another rubber tube connected with another tambour. Hence one is able to register oral and nasal vibrations of air in speaking. The formation of consonants and vowel waves becomes visible. The instrument records the finest changes in articulation, as seen in the initial stages of multiple sclerosis and general paresis.

(*c*) For the control of a tone in singing. This registration in connection with the record of respiration gives evidence of many disturbances of the singing voice.

(*d*) To register the stream of air escaping through mouth and nose in speaking. It measures most exactly the various kinds of rhinolalia in such cases as have been described.

(*e*) It measures intensity, duration, quality and exactness of sounds. This is important for numerous nervous diseases and furnishes a clue to their diagnosis, e. g., epilepsy, Huntington's chorea and bulbar paralysis.

4. The otophone. When put before the patient's mouth it indicates his kind of abnormal articulation in lisping.

5. The nasophone. It makes audible the stream of air escaping through the nose in rhinolalia.

6. The phonograph recording machine. It serves as a control of diagnosis and therapy. The record is a permanent diagnostic means not unlike a roentgenogram. It permits reexamining a speech or voice symptom as often as necessary. It makes the patient aware of his speaking technic. Moreover, it is a useful device for students, as they can study from it the symptoms of neurologic conditions which otherwise would hardly be available for them.

7. A baby piano or a little harmonium. It helps to classify the pitch of a sound either spoken or sung; e. g., in cases of functional irritation of the larynx in consequence of too low or too high a speaking voice a new natural pitch has to be determined. The patient accommodates his pitch to the one given by the instrument.

8. The oscillograph. It is not necessary, because of its more technical purposes. Its practical value for diagnosis and therapy is not considerable, and it is expensive.

This is the mechanical outfit. The main instrument, however, has not been mentioned yet: the doctor's ear. This ear should be especially good, trained and—if possible—musical. Its sensitivity must be trained just as the student must acquaint himself with the use of a stethoscope. The roentgenologist's eye sees more on a roentgenogram than the lay person's. The phonetician's eye has to learn to decipher kymographic records. Time and experience will teach one how to deal with the more technical problems.

However, there is something more in the work than the skilful mechanical application of the various apparatus. When dealing with neurotic persons, with those presenting problems of behavior, with children with different intelligence quotients or with parents from the psychiatric ward a special training in psychology and psychiatry is required.

VOICE

Medical phonetics includes study of the organic as well as the functional vocal diseases and of vocal hygiene in schools. Important among organic diseases is singer's nodes. It is known that it does not help only to remove them, because they regrow, unless the cause for their existence, the wrong use of the voice, is removed.

If the shoe presses one must not only cut the corn but also widen the shoe. With the technical means mentioned it is possible to trace and register the incoordinations and to try to correct them.

Many vocal disturbances in all stages of life are results of mutation, or change, of voice. Boys and girls can reveal a prolonged mutation, boys a persistent high pitch—the so-called persistent falsetto voice—without any endocrinologic impairment. Other symptoms of this complex are hoarseness, aphonia and divergence of the chest and head register. Menstruation, pregnancy and senility are likely to cause vocal impairments.

Paralysis of the recurrent nerve can be due to numerous intrathoracic processes, such as gumma, sarcoma, pneumonoconiosis and aneurysm, which can hardly be influenced. After thyroidectomy 3 per cent of all patients suffer from vocal defects caused by a lesion of the recurrent nerve. These defects can be treated successfully with phonetic procedures.

Laryngectomy, laryngofissure and resection of the vocal cords have presented new tasks for the phonetician. The esophagus and the stomach have to replace the lungs. The laryngectomized patient has to learn to swallow air, in order to develop eructation, first into a single sound and in the end into a sentence. Some patients without a larynx even learn to sing.

The functional vocal diseases are often professional. Phonastheny (weakness of voice) with its symptoms of hoarseness, tiredness and nervousness is found in singers as well as speakers. If laryngitis is wrongly diagnosed and the vocal cords are treated—or rather mistreated—with silver nitrate or if one advises the patient to take a vacation and rest his voice, he will come back, his voice rested, and start his professional talking with the same incorrect technic as before. Some weeks later the same trouble will start again, and neither silver nitrate, nor short waves nor vitamin B will give him any relief. The method of choice is phonetic examination and instruction in the physiologic use of the voice.

The impairments of children's voices in schools is due to choral singing. Each child tries to excel his neighbour in intensity of voice. Another cause is the glottic stroke (hard attack of a sound instead of a soft one). To prevent the children from enforcing an exaggerated range in singing the songbooks nowadays are adapted to the physiologically small range of a child. Nevertheless they sing operettas in public schools which surpass their natural capacity.

Singing during mutation is another problem, especially in schools with children of different races. Mutation endangers the larynx and voice and constitutes the basis of many vocal diseases. The handling of this problem in schools is difficult, because, according to their race, children undergo mutation between 9 and 15 years of age. During this time, then, one group of children needs special vocal care.

SPEECH

Speech problems occur more frequently in clinics than voice problems. Many patients belong to the group with pathologic speech development. The first thirty months of life are considered a period of physiologic mutism. If a child between the ages of 2½ and 3 years does not talk, this deficiency is called "prolonged physiologic mutism." If it persists one speaks of "hearing-mutism." This term signifies the lack of a speaking function *in spite of* normal hearing. This is in contrast with deaf-mutism, which signifies the lack of a speaking function *because* of deafness. Hearing-mutism takes normal intelligence for granted—otherwise, the phenomenon is called idiotic-mutism.

Word deafness and mind deafness should be included in this chapter. Both are based on an existing hearing function, which, however, cannot be utilized on account of an assumed anatomic or psychologic lesion. To know these facts is especially valuable for the interpretation of hearing tests. Completely negative responses to a test in a child need not indicate real deafness. It could also be brought about by mind deafness (i. e., the incapability of interpreting noise, sounds and words).

A test often valuable for the differential diagnosis between hearing-mutism and deaf-mutism is Fröschels' "itching reflex," a lid reflex to scratching of the auditory canal, positive with hearing-mutism, negative with deaf-mutism.

The therapy has to stimulate both acoustic concentration and acoustic perception. The value of clipping the frenum of the tongue in order to accelerate the development of speech is only imaginary. The theory of "tonguetie" goes back to Celsus (30 A. D.), to whom the tongue was the speech center. Since brain centers are known it has become ridiculous to try to influence the psychologic factor of the development of speech by applying a pair of scissors.

Functional disorders of speech consist of mispronunciations, non-pronunciations or substituting one letter for another. Lisping can be interdental, addental, lateral or nasal. It is due hardly ever to an anatomic basis (gap between teeth, etc.) but to psychologic reasons or to general articulatory awkwardness.

Stutterers crowd the speech clinics. Their problem has gained popular interest because of the publicity in magazine and newspaper articles. The advantage of these is undoubtedly that the stutterer no longer seems ridiculous. Stuttering being a complex of psychoneurotic and psychologic manifestations it is absolutely necessary that doctors should diagnose the patients. Textbooks of otology contain quotations from psychologists on the nature of stuttering and its neurologic nature, as if medical phonetics did not exist. And the continued use of the terms "stuttering" and "stammering" without any scientific discrimination proves sufficiently the lay character of many publications. To a physician, the repetition of letters or syllables is the manifestation of a clonic spasm, and the pressure symptom, when the patient is unable to start a sound, the manifestation of a tonic spasm.

Stuttering can be explained as the expression of an exaggerated pathologic concentration on the details of speech mechanism. It leads to superfluous muscular strain.

The dogmatic quarrel about the origin of stuttering is only theoretic. The importance of left-handedness has been greatly overestimated. The problem of handedness is by no means new, but has been well known for forty-five years. It is of some significance for about 6 per

cent of all stutterers. In many other cases the condition has to be determined as "associative aphasia," aphasia due to wrong associations of "I cannot speak" or "It does not work." An assumed organic inferiority leads to the term spastic neurosis of coordinations if one assumes a special center for the coordination of syllables.

Another problem closely related to stuttering is paraphrasia praeceps, cluttering, a precipitancy of speech with marked rhythmic impairments. This speech disorder is closely connected with a corresponding disorder of the thinking process.

The otologist is more interested in organic speech defects, e. g., impairments of the soft palate. Open rhinolalia is a symptom of cleft palate or of paralysis after diphtheria, after encephalitis, in bulbar or pseudobulbar processes or in myasthenia gravis and frequently is purely functional without any anatomic lesions.

The diagnosis can be registered by the apparatus described. The therapy is active (exercises) and passive (electric vibration, massage and faradization).

Closed rhinolalia occurs in cases of adenoids or excessive nasal hypertrophy. Mixed rhinolalia is the coincidence of the symptoms of the open and the closed form.

In phonetics mutism is considered the innate incapability of talking. Any loss of a previously existing power of speech must be called "aphasia." Cases of motor and sensory aphasia in children, as well as in adults, offer material to speech clinics that mostly is badly neglected. Patients who have lost the power of speech (physical mechanism) or the faculty of language (psychologic mechanism) by apoplexy have to face a hopeless life because of their incapability of expressing themselves. The therapy consists in reeducation. Some weeks after the occurrence of apoplexy with motor aphasia the phonetician carefully teaches with the help of a mirror the placement of lips and tongue. With sensory aphasia the demonstration of pictures and objects accompanied by their names slowly pronounced helps to rebuild the language and to repair the vocabulary. A close cooperation between neurologic and phonetic clinics should be established for both therapeutic and diagnostic reasons.

Scripture developed the theory that from analyzing records of speech on the kymograph one can establish the diagnosis of multiple sclerosis, of epilepsy and of other nervous diseases, either by counting the vibrations (to find the symptom of the rigid speech melody characteristic of epileptic patients) or by comparing explosions (a patient with dementia paralytica is unable to explode the letter "p" regularly if he repeats it several times).

CONCLUSION

The more exact methods of modern experimental phonetics, which before have been used only for linguistic purposes, will gradually find their way into the otologist's office. The movements of the larynx, of the palate and of the lips, as well as any air vibrations of voice and speech, can be registered with the same exactness as the electrocardiogram records the auricular and ventricular activity of the heart.

It must be admitted that a great number of the nervous diseases mentioned do not, strictly speaking, belong to the otologist's field. However, these diseases reveal symptoms in the organs of his field, which can only be detected by a thoroughly trained otologist. Parkinson's paralysis, for instance, affects the voice, and one can help to establish a diagnosis by recording the inaudible microscopic tremulous movements of the larynx. Specialists interested in constitutional problems will find that the human voice gives new evidence and furnishes new clues for certain constitutional types. To the different constitutional types (Sigaud, Kretschmer) are correlated different variations of voice. Thus phonetics is able to spread the importance of otorhinolaryngology and may be considered its fruit-bearing branch.

OTOSCLEROSIS

POSTAURICULAR APPROACH IN OPERATIVE TREATMENT

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CINCINNATI

Operative treatment of otosclerosis is attracting considerable interest, but the operative technic by the endaural route as well as by the posterior approach has been so trying and exhausting, the operation often lasting from three to five hours or more, that many aurists have been discouraged from attempting to settle its value for themselves.

Whether operative treatment of the disease will be generally accepted will be known only after more extensive use and experimentation. Canfield [1] is especially to be commended on his recent experiments with labyrinthine fistulas.

I wish to describe a postauricular approach with little or no disturbance of the middle ear, which is reasonably simple to perform, giving an excellent opening in the horizontal semicircular canal by means of an instrument which gives greater ease of control than a gouge or a chisel and which I believe will be more acceptable to most operators than an electric burr. This operation also offers a method, which I shall describe in detail, of securing the thinnest and most approximate membrane that could reasonably be obtained to cover the opening made in the canal.

If local anesthesia is to be used, the patient is given 1½ grains (0.1 Gm.) of seconal [2] on the evening before operation. Two hours preceding operation 3 grains (0.2 Gm.) of sodium amytal is given by mouth, with a hypodermic injection of ¼ grain (0.02 Gm.) of morphine and 1/200 grain (0.3 mg.) of scopolamine hydrobromide. One hour before operation a second hypodermic of ⅙ grain (0.01 Gm.) of morphine is given.

From the Otolaryngological Department, College of Medicine, University of Cincinnati.

1. Canfield, N.: Labyrinthine Fistulas: Report of Experiments on the Vital Response to Various Methods of Producing Defects in Bone, Arch. Otolaryng. **30**: 50-62 (July) 1939.

2. Seconal is stated by the manufacturer to be sodium propylmethylcarbinylallylbarbiturate. The preparation has not been considered by the Council on Pharmacy and Chemistry of the American Medical Association.

Locally, a 1 per cent solution of procaine hydrochloride with epinephrine is injected through the skin and the periosteum in the line of incision and along the posterior bony wall of the canal.

Local anesthesia has been recommended for the endaural approach because of the unusually long time of operation and the hope of a response from the patient when the semicircular canal is opened. For the postauricular approach, a much shorter operation, I see no reason why a general anesthetic should not be used if preferred.

With the area under local anesthesia the patient is usually so stuporous that any indication of better hearing, unless volunteered without questioning at the time of the uncapping of the semicircular canal, is not significant.

The incision is carried well posteriorly in the edge of the hair line and not below the tip of the mastoid. The tissues, including the periosteum, are elevated forward, the superior and posterior bony walls of the auditory meatus being exposed.

The cortex is removed by means of a Hudson hand perforator and a burr,[3] the posterior wall of the canal being lowered as the burr deepens the mastoid cavity. The horizontal semicircular canal and the short limb of the incus are brought into view by means of a Spratt curet and a round one.[3]

The soft tissues are carefully separated from the outer portion of the posterior bony wall of the canal, the operator being careful to remove the bone above and below to give the full exposure of the soft tissues which cover the posterior wall. Great care must be taken that the deeper tissues near the posterior tympanum are not disturbed or separated at this time.

Once the posterior soft tissues are exposed in the outer two thirds, a narrow scalpel is used to incise the soft tissues of the posterior wall of the canal through and through at right angles (fig. 1) to the canal 1 cm. from the posterior attachment of the tympanum.

Now the outer part of the canal can be moved forward (fig. 1 *C*) without danger of any detachment at the annulus tympanicus, a most important precaution.

The rest of the bony posterior wall of the canal is slowly removed from the soft tissues by means of John D. Richards' mastoid curets (fig. 2), which must be perfectly sharp and small or moderate in size. The bone is thinned anteriorly and lowered by shaving from above downward, the upper edge of the bony mastoid cavity being used as a fulcrum (fig. 3).

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3. Goodyear, H. M.: The Radical Mastoid Operation: Use of the Tensor Tympani Muscle in Closing the Eustachian Tube, Laryngoscope **49**:580-590 (July) 1939.

The posterior bony wall of the auditory canal is lowered to approximately the level of the annulus tympanicus.

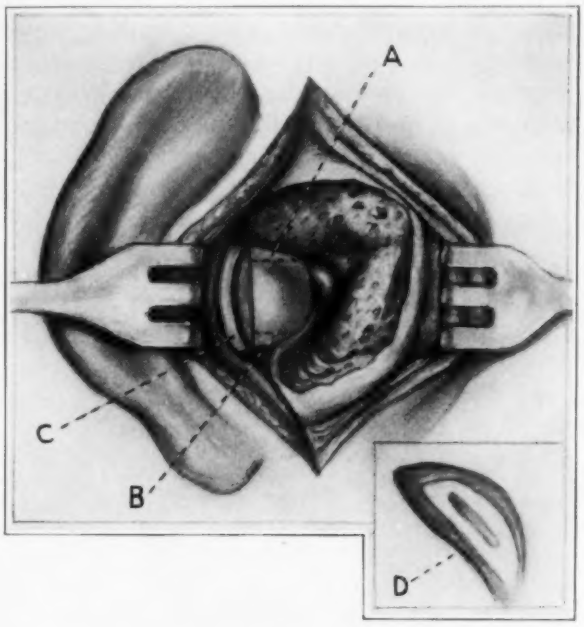

Fig. 1.—Transverse incision and vertical incisions, *A* and *B*, outlining the skin flap. *C*, indicates the freed portion of the auditory canal; *D*, the opening in the external canal.

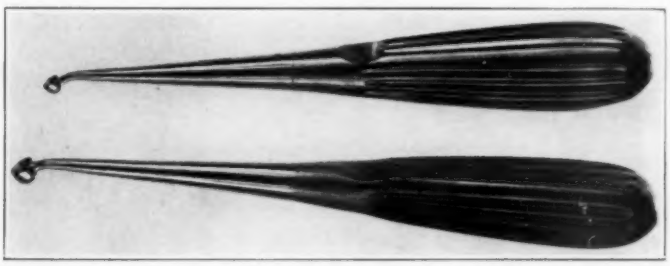

Fig. 2.—John D. Richards' mastoid curets, bent at an angle of 45 degrees, sizes 0 and 2 (Tieman & Co., New York).

With the horizontal semicircular wall of the canal exposed, it is uncapped with a small sharp Richards curet, the operator slowly shaving downward and slightly posteriorly, parallel to the canal, using the upper edge of the mastoid cavity as a fulcrum (fig. 3).

First, a small bluish gray line appears, representing the bony canal, which is outlined for a distance of 3 to 5 mm. The exposure is made the full width of the labyrinthine canal (fig. 1 *D*), and the endosteum is broken, some perilymphatic fluid being permitted to escape. With a magnifying glass the intact membranous labyrinth may be seen.

Vertical incisions (fig. 1 *A* and *B*) are now made in the skin flap, the operator guarding against any pulling that might separate it from the tympanum. This flap is then turned backward and comes to lie firmly over the opening in the horizontal canal (fig. 4).

A small piece of gauze treated with petrolatum or paraffin is placed over the skin flaps and is left in place for eight to twelve days.

Fig. 3.—Upper edge of the mastoid cavity being used as a fulcrum for a Richards curet.

A circular flap (fig. 5) is cut to enlarge the external auditory canal and is thinned, turned upward and backward and sutured to the periosteum, the fascia or the edge of the anterior skin flap.

The cavities in the mastoid and the ear are gently packed with petrolatum gauze, and the postauricular wound is completely closed.

The packing is not disturbed for seven days, when it is partly removed. It is completely removed by the eighth or twelfth day.

I am not overenthusiastic about the operative treatment of otosclerosis, and it is only by encouraging simpler operative procedures that any great interest and experimentation can be expected in the field.

Jenkins,[4] in his work, opened the horizontal canal twice for a distance of 1.5 mm.; once he left the opening uncovered, and once he covered it with skin from the posterior meatal wall. I have had in my possession from the medical library at Washington a description of his operation, done only once in his career, and no detail of what sort of skin flap he used or how he used it was given. Of course, the opening he made in the canal was insignificant.

I have been unable to find any detailed description in the literature of the proper preparation of a skin flap from the posterior wall of the auditory canal through a postauricular opening that is suitable for this work. The horizontal incision across the soft tissues, which I have proposed, is of paramount importance. Its use along with the Richards curet for the removal of the bone from the soft tissues and for the opening of the horizontal semicircular canal cannot be fully appreciated except in the actual preparation of this exceedingly delicate tissue flap.

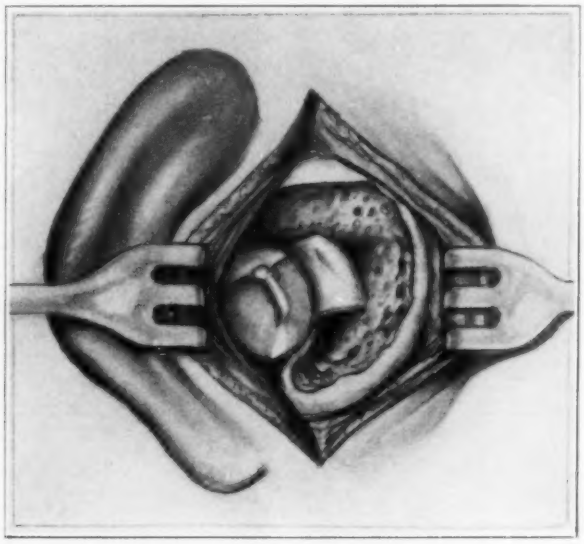

Fig. 4.—Flap turned backward and placed over the opening in the external semicircular canal.

I shall quote from Lempert:[5]

> The cutaneous lining, which forms the meatal portion of the tympanomeatal membrane and unites this portion with the tympanic portion by extending to

4. Jenkins, G. J.: Otosclerosis: Certain Clinical Features and Experimental Operative Procedures, Transactions of the International Congress of Medicine (1913), London, 1914, sect. 16, pt. 2, pp. 609-618.

5. Lampert, J.: Improvement of Hearing in Cases of Otosclerosis, Arch. Otolaryng. **28**:42-97 (July) 1938.

form the delicate outer cutaneous layer of the tympanic membrane, is very thin. In its extent outward from the margin of the membrana tympani for a distance of about 10 mm. the thickness of the meatal portion varies from 0.01 to 0.03 mm. The tympanomeatal membrane, when completed, is the thinnest cutaneous membrane that could possibly be obtained plastically or grown biologically. It is destined to seal the perilymph space through the window which is to be created in the bony capsule of the external semicircular canal.

I have described a method of obtaining this cutaneous tympanomeatal membrane completely intact, without injury, without severing of the meatal portion from its attachment to the tympanic portion and without perforation of the tympanic membrane, as Lempert [5] specifies as a part of his technic.

Fig. 5.—Outline of circular meatal skin flap.

Holmgren,[6] by the posterior approach, covered the canal openings with gold leaf, fat, a skin graft, the dura and the mucoperiosteum of the ampulla.

Sourdille [7] performs what he terms a tympanolabyrinthopexy in three principal stages at intervals of four or five months.

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6. Holmgren, G.: The Surgery of Otosclerosis, Ann. Otol., Rhin. & Laryng. **46**:1-12 (March) 1937.

7. Sourdille, M.: New Technique in the Surgical Treatment of Severe and Progressive Deafness from Otosclerosis, Bull. New York Acad. Med. **13**:673-691 (Dec.) 1937.

My work has been done on cadavers. I have used the skin flap twice on the living, once in a modified radical mastoidectomy and a second time to cover an opening which I made in the horizontal canal. The second patient now shows horizontal nystagmus with dizziness on the application of light air pressure to the external auditory canal. Thus, the fistula after three and one half months is still open and highly sensitive to the fistula test.

Lempert [5] stated, "I believe that it is reasonable to assume that a fistula which has not shown signs of beginning regeneration of bone after two months will remain permanently open."

The middle ear is completely sealed off in both my cases in which the skin flap was used, yet the head of the malleus was not removed.

556 Doctors' Building.

PROBABLE PATHOGENIC STREPTOCOCCI AND STAPHYLOCOCCI IN CHRONIC LOW GRADE ILLNESS

AN ANALYSIS OF THEIR FREQUENCY IN THREE HUNDRED AND NINETY-FIVE CASES

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Recently described in vitro tests of bacteria [1] have been shown to give results parallel with certain pathogenic properties of the cultures and with other biologic findings.[10] Although cultures reacting positively to these tests are probably pathogenic,[10] for clarity they will be referred

1. (*a*) Chapman, G. H.; Berens, C.; Peters, A., and Curcio, L.: Coagulase and Hemolysin Tests as Measures of the Pathogenicity of Staphylococci, J. Bact. **28**:343-363, 1934. (*b*) Chapman, G. H., and Berens, C.: Crystal Violet Agar as a Differential Medium for Staphylococci, ibid. **29**:437-448, 1935. (*c*) Chapman, G. H.: Specificity of the Dye in the Crystal-Violet-Agar Reaction of Staphylococci, ibid. **32**:199-205, 1936. (*d*) Chapman, G. H.; Lieb, C. W.; Berens, C., and Curcio, L.: The Isolation of Probable Pathogenic Staphylococci, ibid. **33**:533-543, 1937. (*e*) Chapman, G. H.; Lieb, C. W., and Curcio, L.: The Use of Bromthymol Blue Agar and Phenol Red Mannitol Agar for the Isolation of Pathogenic Types of Staphylococci, Am. J. Clin. Path. **8**:3-11, 1938. (*f*) Chapman, G. H.; Berens, C.; Nilson, E. L., and Curcio, L. G.: The Differentiation of Pathogenic Staphylococci from Non-Pathogenic Types, J. Bact. **35**:311-334, 1938. (*g*) Rawls, W. B., and Chapman, G. H.: Experimental Arthritis in Rabbits, J. Lab. & Clin. Med. **21**: 49-64, 1935. (*h*) Chapman, G. H., and Rawls, W. B.: Studies of Streptococci: I. Qualitative Differences in Resistance to Various Agents, J. Bact. **31**:323-331, 1936. (*i*) Chapman, G. H., and Curcio, L.: Studies of Streptococci: II. Quantitative Differences in Resistance to Sodium Bicarbonate and Hexylresorcinol, ibid. **31**:333-337, 1936. (*j*) Chapman, G. H.; Berens, C., and Nilson, E. L.: Studies of Streptococci: III. Preliminary Attempts to Correlate Resistance to Chemicals, etc., with Pathogenic Effects, ibid. **31**:339-346, 1936. (*k*) Chapman, G. H.: Studies of Streptococci: IV. Resistance of Enterococci, ibid. **32**:41-46, 1936. (*l*) Chapman, G. H.; Stiles, M. H., and Berens, C.: The Isolation and in Vitro Testing of Pathogenic Types of Non-Exotoxic Streptococci, Am. J. Clin. Path. **9**:20-27, 1939. (*m*) Stiles, M. H., and Chapman, G. H.: Relationship Between Agglutinability and Certain in Vitro Tests of Staphylococci, Streptococci and Colon

(Footnote continued on next page)

to as "in vitro positive" rather than "probable pathogenic." The development of these tests has made possible the study of the relation of large numbers of staphylococci and streptococci associated with chronic, and sometimes with acute, illnesses. In the investigation to be discussed the methods were used in the study of the distribution of probable pathogenic staphylococci and streptococci in patients with low grade chronic illness. Observations on the distribution of the sedimentation rates and nonfilament-filament ratios of 292 of these patients have been reported.[2]

MATERIAL

Of the 395 patients studied, most of them showed more than one disease, 96.3 per cent had clinical evidence of involvement of the nose or nasopharynx; 52.2 per cent had classic evidence of chronic sinusitis, and 44.1 per cent had symptoms of the condition which has been called "chronic nasopharyngitis" by Burbank [3] and "sinusitis" by Chase.[4] There were 31 cases of tonsillitis and 17 of other types of oral infection. (Most of the patients had been subjected to tonsillectomy and had received satisfactory dental care.) Bronchitis was present in 83 cases, colitis or irritability of the colon in 67, recurrent or chronic gastroenteritis in 89, symptoms referable to the biliary tract in 82, inflammatory conditions of the urinary tract in 38, rheumatoid arthritis in 16 and other forms of arthritis in 38. A group of 52 patients, who had symptoms which have been called "prodromes to arthritis" [5] were classified as having "rheumatism."

Of these conditions, which are more or less generally considered secondary to infectious processes, at least one was present in each patient studied. In addition, in the group there were 22 patients with asthma, 18 with duodenal ulcer and 22 with migraine.

The patients were classified according to the severity of illness as having symptoms which were mild or recurrent, moderate, severe or acutely exacerbated.

METHODS

Cultures of material from the nasal and oral cavities were made for all subjects. Cultures of the feces were made for all but a small number.

Bacilli Isolated from Persons Suspected of Having Chronic Infection, J. Lab. & Clin. Med. **24**:620-623, 1939. (*n*) Chapman, G. H., and Berens, C.: Comparison of Intradermal Tests with Agglutinability and Certain in Vitro Tests of Streptococci, Staphylococci, Micrococcus Catarrhalis and Colin Bacilli Isolated from Persons Suspected of Having Chronic Infection, ibid. **24**:601-604, 1939. (*o*) Chapman, G. H.; Berens, C.; Lieb, C. W.; Rawls, W. B., and Stiles, M. H.: Examination of Cultures from Persons Suspected of Having Chronic Infection, Am. J. Clin. Path. **9**:491-503, 1939.

2. Stiles, M. H.: Sedimentation Rate and Nonfilament-Filament Ratio in Low-Grade Chronic Illness, Arch. Int. Med. **63**:664-678 (April) 1939.

3. Burbank, R.: Etiology and Treatment of Chronic Arthritis, J. A. M. A. **99**:1489-1494 (Oct. 29) 1932.

4. Chase, W. D.: Etiology and Diagnosis of Sinusitis, Pennsylvania M. J. **39**:389-395, 1936.

5. Hench, P. S.; Bauer, W.; Fletcher, A. A.; Ghrist, D.; Hall, F., and White, P.: The Present Status of the Problem of "Rheumatism," Ann. Int. Med. **8**: 1495-1555, 1935.

Because there was no essential difference between cultures of material from the right and from the left naris of a patient, between those of material from the right and from the left tonsil or between those of material from the tonsils and pharynx, indicating intermixing of the flora throughout a rather wide area, the cultures of material from these parts are listed as of material from the oral or from the nasal cavity. Material for cultures was taken from the nasal cavity by passing the swab as far back as possible and from the gingival margin, tonsillar fossae, pharynx and nasopharynx. In collecting specimens of feces, patients were given a laxative pill. The soft, mushy specimen was mixed with a little water and put in a test tube. A plug of absorbent cotton was forced through to the bottom of the tube, filtering the specimen. Serial dilutions were plated on blood agar, bacto tryptose agar being used as a base medium.[11]

In vitro positive staphylococci were isolated by the method of Chapman, Lieb, Berens and Curcio.[1d] In vitro positive streptococci were isolated by the method of Chapman, Stiles and Berens.[11]

The methods for the differentiation of pathogenic from nonpathogenic types of staphylococci have been summarized recently.[1f] For simplicity in tabulation, strains that coagulated human or rabbit plasma within twenty-four hours [6] were considered in vitro positive.

Smooth strains of streptococci which were resistant to the "bactericidal" action of fresh, diluted, defibrinated guinea pig blood [7] or were resistant to the action of critical time/dilutions of sodium bicarbonate and hexylresorcinol [8] were considered in vitro positive. Enterococci were disregarded.[9]

Because only in vitro positive staphylococci and streptococci were considered in tabulating the findings, they will be referred to simply as staphylococci or streptococci.

RESULTS

Streptococci.—Of the 395 patients, 98.7 per cent yielded streptococci in at least one of the cultures. They were found most frequently in the oral cavity and least frequently in the nasal cavity (table 1). Alpha types were most common, being present in the oral cavity in 96 per cent of the cases. Beta strains were recovered in 9 per cent (table 2).

Staphylococci.—Staphylococci were found in 69.8 per cent of the 395 patients. They occurred most frequently in the nose and least frequently in the feces. Because most pathogenic staphylococci are of the aureus type it was expected that aureus strains would predominate. However, 18.5 per cent of the patients had nasal strains, and 10.9 per cent had oral strains of the albus variety (table 2).

Frequency.—When the cultural findings were analyzed on the basis of the clinical condition, there was a direct relation between the severity

6. Chapman, Berens, Peters and Curcio.[1a] Chapman, Lieb, Berens and Curcio.[1d] Chapman, Berens, Nilson and Curcio.[1f]

7. Rawls and Chapman.[1g] Chapman and Rawls.[1h] Stiles and Chapman.[1m]

8. Chapman and Rawls.[1h] Chapman and Curcio.[1i] Chapman, Berens and Nilson.[1j] Chapman, Stiles and Berens.[11] Chapman and Berens.[1n]

9. Chapman.[1k] Chapman, Stiles and Berens.[11]

of symptoms and the proportion of patients with streptococci in more than one cultural source (table 3). Those with severe symptoms were more likely to have fecal streptococci in addition to the oral strains. The incidence of nasal streptococci increased to a lesser extent (table 4). There was also a tendency for the patients with severe conditions to

TABLE 1.—*Incidence of in Vitro Positive Streptococci and Staphylococci in the Nose, Oral Cavity and Feces*

Source	Streptococci		Staphylococci	
	Number	Percentage	Number	Percentage
Nose	30	7.6	233	59.0
Oral cavity	384	97.2	195	49.4
Feces	127	32.2	23	5.8

TABLE 2.—*Distribution of Types of in Vitro Positive Streptococci and Staphylococci*

Source	Streptococci						Staphylococci			
	Alpha		Beta		Gamma		Albus		Aureus	
	No.	%	No.	%	No.	%	No.	%	No.	%
Nose	17	4.3	6	1.5	8	2.0	73	18.5	176	44.6
Oral cavity	380	96.2	37	9.4	140	35.4	43	10.9	159	40.3
Feces	107	27.1	7	1.8	46	11.6	6	1.5	18	4.6

TABLE 3.—*Relation Between Severity of Symptoms and Incidence of in Vitro Positive Streptococci and Staphylococci*

Type of Symptoms	No. of Cases	Per Cent of Cases in Which In Vitro Positive Organisms Occurred in More than One Cultural Source	
		Streptococci	Staphylococci
Mild	174	32.2	47.2
Moderate	156	35.2	48.2
Severe	45	48.9	44.5
Acutely exacerbated	20	65.0	45.0

Note.—When staphylococci occurred, they usually were present in both the nasal and the oral cavity, whereas streptococci occurred more frequently in the oral cavity alone. Hence, the percentage of cases in which positive staphylococci occurred in more than one source was relatively high in all groups.

have a richer flora of streptococci. Of the patients with mild symptoms only 10 per cent had abundant streptococci, compared with 16 per cent of those with moderate symptoms, 18 per cent of those with severe symptoms and 30 per cent of those with acutely exacerbated symptoms.

There was no constant relation between the incidence of staphylococci and the clinical severity, although there was a tendency toward a lower incidence in the patients with more severe symptoms (table 3 and 4).

When the frequency of albus and that of aureus strains were compared, the patients with more serious conditions yielded a higher proportion of aureus strains, particularly from the nose.

Patients with acutely exacerbated symptoms had a higher proportion of streptococci, particularly of the beta type, than did those of any of the other groups, and also a higher proportion of Staphylococcus

TABLE 4.—*Relation Between Severity of Symptoms and Incidence of in Vitro Positive Streptococci and Staphylococci in Various Sources*

	Per Cent of Cases in Which In Vitro Positive Organisms Occurred							
	Streptococci				Staphylococci			
Type of Symptoms	Nose	Oral Cavity	Feces	Any Focus	Nose	Oral Cavity	Feces	Any Focus
Mild	4.0	96.6	31.6	98.9	64.4	48.3	5.7	71.2
Moderate	9.6	97.6	28.2	98.1	61.5	51.3	6.4	70.5
Severe	13.3	98.7	37.8	100.0	60.0	42.2	6.7	64.4
Acutely exacerbated	10.0	100.0	55.0	100.0	50.0	60.0	0.0	65.0

TABLE 5.—*Incidence of in Vitro Positive Streptococci and Staphylococci When Observations Were Distributed According to Clinical Diagnosis*

		Per Cent of Cases in Which In Vitro Positive Organisms Occurred				
		Alpha Streptococci		Staphylococci	Staph. Aureus	
Clinical Group	No. of Cases	Nose	Feces	Any Focus	Nose	Oral Cavity
All cases	395	4.3	27.1	69.8	44.5	40.3
Sinusitis	206	5.8	25.2	71.8	43.7	42.7
Nasopharyngitis	174	2.9	31.6	65.5	**44.2**	36.8
Tonsillitis	31	6.5	25.8	83.9	61.3	58.1
Oral infection	17	11.9	29.4	82.3	47.1	41.2
Bronchitis	83	6.0	21.3	78.7	54.2	45.8
Colitis	67	4.5	**47.8**	65.6	43.3	43.3
Biliary tract symptoms	82	1.2	33.0	70.7	45.2	41.5
Rheumatoid arthritis	16	0.0	37.5	56.3	43.7	18.8
"Other" arthritis	38	2.6	23.7	**50.0**	39.5	31.6
"Rheumatism"	52	3.9	38.5	75.0	51.9	42.3
Asthma	22	4.5	**54.6**	77.3	45.4	40.9
Duodenal ulcer	18	5.6	33.3	55.6	44.4	16.7

Note.—Because other groups of organisms showed little variation, only alpha streptococci in nose and feces, total staphylococci, and Staph. aureus in nose and oral cavity are tabulated.

The percentages in bold face type are statistically significant, as mentioned in the text.

aureus in the nose and throat. However, the number of cases was too small for statistical significance.

When the bacteriologic findings were distributed according to the disease (table 5), a number of variations were found. Patients with sinusitis had a richer flora of streptococci and staphylococci in the nose and throat and a higher incidence of staphylococci but fewer fecal streptococci and staphylococci than did those with nasopharyngitis.

Staphylococcus aureus was abundant in the noses and throats of patients with tonsillitis. Those with bronchitis yielded profuse growths of nasal and oral staphylococci.

Patients with colitis had a high incidence of fecal streptococci and Staphylococcus aureus. Streptococci were present in the feces in 58.2 per cent of the cases, staphylococci in 12 per cent and either or both organisms in 64.2 per cent. These values were significantly above the average.

In patients with arthritis the incidence of staphylococci was low; they were present in only 56.3 per cent of the patients with rheumatoid and 50 per cent of those with "other" arthritis. More than the average number of patients with rheumatoid arthritis yielded fecal streptococci, while with "other arthritis," mostly osteoarthritis, the number was less than average. With "rheumatism" the incidence of fecal streptococci and of oral and nasal strains of Staphylococcus aureus was greater than average.

In cases of asthma the incidence of streptococci was high, the proportion of patients with alpha and gamma streptococci in the feces and of those with gamma streptococci in the nose and throat being higher than in any other group.

COMMENT

The incidence of streptococci reacting positively to resistance tests was so high that it may be assumed that in vitro positive (possible pathogenic) streptococci are practically universally present in persons with low grade chronic illness. The finding of increased numbers of in vitro positive streptococci in patients with severe symptoms suggests that these organisms may be significant. That such streptococci may be of pathologic importance was suggested by Rawls and Chapman's [18] observation that bactericidin-positive strains are capable of producing arthritis in rabbits, while other strains either are incapable of producing arthritis or else require larger doses of culture, a longer time and a larger number of injections. Further, testing streptococci by animal injection, Chapman, Berens and Nilson [19] found that in vitro positive strains produced lethal effects in 84.9 per cent of 33 rabbits, while in vitro negative strains produced only 1 death (of questionable significance because it was apparently due to factors other than the pathogenicity of the strain) in 10 rabbits.

If it is assumed that pathogenic streptococci are present in practically all persons with chronic illness and that they may be present in apparently healthy persons, the question arises why these persons should exhibit such varying degrees of health. Varying response to the presence of pathogenic streptococci might be conditioned by noninfectious factors, by the number and the degree of pathogenicity of the streptococci present,

by the number of areas infected and by varying degrees of sensitization to streptococci. The role of sensitization, which has been demonstrated by Baker, Thomas and Penick [10] in the production of experimental carditis in rabbits, has been well reviewed [11] and has been discussed editorially in the *Journal of the American Medical Association.*[12]

While the incidence of streptococci in different clinical conditions varied considerably, the variations were statistically significant only in relation to changes in the severity of symptoms and in cases of colitis and of asthma.

As shown in a previous paper,[2] patients with severe symptoms had higher nonfilament-filament ratios (nonfilament-filament ratio = $\frac{\text{nonfilamented neutrophils} \times 100}{\text{filamented neutrophils}}$) and sedimentation rates than did other patients, with the exception that patients whose symptoms were acutely exacerbated did not have as high sedimentation rates as those with symptoms

TABLE 6.—*Correlation Between Severity of Symptoms, Incidence of in Vitro Positive Streptococci, Nonfilament-Filament Ratio and Sedimentation Rate*

Type of Symptoms	Per Cent of Cases in Which In Vitro Positive Streptococci Occurred in More Than One Cultural Source	Mean Nonfilament-Filament Ratio*	Mean Sedimentation Rate, Mm. in 1 Hour (Westergren)
Mild	32.2	18.56	7.20
Moderate	35.2	24.22	19.99
Severe	48.9	40.10	39.90
Acutely exacerbated	65.0	44.00	9.50

* Nonfilament-filament ratio = $\frac{\text{Nonfilamented neutrophils} \times 100}{\text{Filamented neutrophils}}$

of longer standing. The incidence of in vitro positive streptococci also paralleled the severity of symptoms and hence the nonfilament-filament ratio and the sedimentation rate (table 6).

The findings in regard to pathogenic types of staphylococci were consistent with those of Hallman,[13] who used the coagulase test, pigment

10. Baker, B. M.; Thomas, C. B., and Penick, R. M., Jr.: Experimental Carditis: Changes in the Myocardium and Pericardium of Rabbits Sensitized to Streptococci, J. Clin. Investigation **14**:465-473, 1935.

11. Rackemann, F. M.: Allergy: A Review of Current Literature, Arch. Int. Med. **55**:141-167 (Jan.) 1935. Hitchcock, C. H.: Tissue Reactivity to Streptococci and Its Bearing upon the Problem of Arthritis, New York State J. Med. **34**: 1022-1027, 1934. Hench, P. S.; Bauer, W.; Ghrist, D.; Hall, F.; Holbrook, W. P.; Key, J. A., and Slocumb, C. H.: The Present Status of Rheumatism and Arthritis, Ann. Int. Med. **11**:1089-1247, 1938.

12. Allergic Tissue Cultures, editorial, J. A. M. A. **108**:2044-2045 (June 12) 1937.

13. Hallman, J. A.: Pathogenic Staphylococci in the Anterior Nares: Their Incidence and Differentiation, Proc. Soc. Exper. Biol. & Med. **36**:789-794, 1937.

production, hemolysis, mannitol fermentation and the crystal violet agar reaction, and of McFarlan,[14] who used hemolysin production, the coagulase test and mannitol fermentation, in studying students and hospital patients.

While the pathologic significance of in vitro positive staphylococcic strains is more generally recognized, staphylococci, particularly albus strains, appear to have less significance than streptococci according to the observations reported here. There was considerable variation in the incidence of staphylococci in different clinical conditions, but with the probable exception of the lower incidence in arthritis other than rheumatoid the variations were not statistically significant.

The low incidence of both staphylococci and streptococci in osteoarthritis is consistent with the general belief that in this form of arthritis factors other than infection may be of considerable importance.

SUMMARY

Cultures were made on 395 patients with low grade illness, to determine the incidence of streptococci and staphylococci reacting positively to certain in vitro tests. These tests had previously been shown to have given results parallel with certain pathogenic properties of the cultures and were, therefore, considered useful as indicators of probable pathogenicity in an extensive investigation of this type. Adequate animal inoculation tests would have been impractical.

In vitro positive streptococci were found in the oral cavities of practically all the patients. Alpha strains of in vitro positive streptococci occurred in the oral cavity in 96 per cent, beta strains in 9 per cent and gamma strains in 35 per cent of the patients. Fecal cultures were the next most frequent source of streptococci, alpha strains occurring in 27 per cent, beta in 2 per cent and gamma in 12 per cent. In vitro positive nasal streptococci were comparatively infrequent, suggesting that nasal secretions may inhibit the growth of such organisms.

Patients with severe or acutely exacerbated symptoms had more abundant in vitro positive streptococci, present in more cultural sources, than did patients with mild or moderate symptoms. Patients with colitis and asthma had in vitro positive streptococci in more sources than did patients with other diseases.

In vitro positive staphylococci were found in approximately 70 per cent of the patients. They were present in 60 per cent of the nasal cultures and in 50 per cent of the cultures of material from the oral cavity but in only 6 per cent of the fecal cultures. There was a tendency for

14. McFarlan, A. M.: Incidence of Pathogenic Staphylococci in the Nose, Brit. M. J. **2**:939-942, 1938.

in vitro positive strains of staphylococci to occur less frequently in serious conditions, particularly in arthritis, and to occur more frequently than average in sinusitis, tonsillitis and bronchitis, but the variations were of probable statistical significance only for arthritis.

CONCLUSIONS

Streptococci reacting positively to in vitro tests which give results parallel with certain pathogenic properties of the cultures were almost universally present in the nasal or oral cavities of a group of 395 patients with low grade chronic illness. The incidence of such streptococci varied with the clinical condition, being significantly greater with colitis and asthma and in cases in which the symptoms were severe or acutely exacerbated.

In vitro positive (probable pathogenic) staphylococci were present in approximately 70 per cent of patients with low grade chronic illness. No significant relation could be found between the incidence of such staphylococci and variations in the clinical condition.

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THE NEXT STEP IN AUDITORY RESEARCH

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CHICAGO

In preparing a discussion of the challenging theme of this symposium, we were guided by an old Greek dictum:

"The rudiments must be strongest."

In the realm of auditory research a multitude of complicated acoustic phenomena is being studied and analyzed for which adequate knowledge of the fundamental principles is not yet available. Therefore, in naming a most desirable "next step in auditory research," a better understanding of the cochlea and its mechanical functioning is chosen without much hesitation. This paper attempts to describe experimental possibilities for the study of the function of the cochlea.

It is evident that the first stages in the process of hearing are physical, the transmission of acoustic energy from the external medium to the nerve endings in the cochlea. It is common to identify the anatomic term "middle ear" with the functional meaning "sound conduction apparatus." Similarly "inner ear" and "sound perception apparatus" are frequently considered synonymous. One should, however, not be deceived by anatomic classification. Certain parts of the inner ear definitely belong to the sound conduction apparatus. The attempt is being made here to describe the mechanical phenomena which occur after the sound enters the inner ear. The investigation can be paradoxically termed "a study of the conduction of sound in the inner ear." The following structures of the inner ear which are important for the mechanism of cochlear stimulation will be considered in this paper:

1. The oval window
2. The round window
3. The intralabyrinthine fluid
   (*a*) The perilymph
   (*b*) The endolymph

From the Department of Surgery of the University of Chicago.

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4. The ligaments of the inner ear
5. The craniolabyrinthine communications
   (*a*) The aquaeductus
   (*b*) The blood vessels
   (*c*) The lymph spaces
   (*d*) The internal auditory meatus

It is obvious that not all these structures actually convey the acoustic energy; they do, however, exert an important influence on the conditions of sound conduction in the inner ear.

These anatomic structures are associated with the following functional problems to be dealt with:

The oval window The round window	1. The windows as portals of entry for the sound 2. Hearing in a windowless cochlea 3. Hearing in a cochlea with one window
The intralabyrinthine fluid	1. Hearing under disturbed hydrostatic conditions 2. The intralabyrinthine fluid as a part of the sound conduction system
The ligaments of the inner ear	Dynamic compressibility of the "soft tissues" of the cochlea
The craniolabyrinthine communications	1. Static conditions; the question of "glaucoma labyrinthi" 2. Half static conditions; the canalicular resistance as a function of frequency; limit of hearing for lower tones and sensitivity of the ear in the condition 3. Dynamic (i. e., acoustic) conditions

These problems were studied in experiments on animals. Our method for objective tests of cochlear function consisted in the observation of the reflex of the acoustic tensor and stapedius muscles. The reflex is a reliable indicator of cochlear function as long as its natural limitations are known and respected.[1] In addition the reflex offers no theoretic difficulties compared with the cochlear potentials or the nerve potentials. Finally, the experiments described in this paper deal with problems which require the inner ear to be opened surgically and functional tests to be performed on a partially opened cochlea. Former tests revealed that the electrical potentials in an opened cochlea behave strangely and seem not to be a true indicator of cochlear function.

The method of conditioned reflexes has shown admirable results. However, for executing experiments a staff of well trained experimenters and an especially equipped laboratory is indispensable. For this reason reliable work on acoustically conditioned animals has been, and can be, done at only a few laboratories.

The objective method of testing function with the acoustic reflex contractions of the muscles of the middle ear differs from the conditioned reflex in that only the lower parts of the acoustic tracts are

1. Kobrak, H.; Lindsay, J. R., and Perlman, H. B.: Value of the Middle Ear Muscle Reflex as Indicator of Hearing, Arch. Otolaryng. **21**:663 (June) 1935.

involved in the reflex of the tensor and stapedius muscles. The higher centers and the cortex are not included. The observations on the reflex of the tensor or stapedius muscles require no special training so far as the animal is concerned. Any experimenter familiar with otologic technic is able to do this type of research without further training.

THE PORTALS OF ENTRY OF SOUND

Our experimental method has permitted conclusive tests in the animal as to whether sound can enter the inner ear through the round window. In the cat a spacious round window niche permits the introduction of a glass tube into direct proximity with the membrane of the round window. A tube was inserted in the round window niche and sealed. Sound was conveyed through tubes directly into the niche. The stimulation of the cochlea through the round window is suprisingly effective.

This experiment demonstrates that the acoustic energy may enter the cochlea through the round window and may reach the nerve endings through the scala tympani and not, as in the case of ossicular sound conduction, by the way of the scala vestibuli. This phenomenon may be called *reversed sound conduction in the inner ear.*

Experiments in which a similar tube was led into the region of the oval window did not give clear results, probably because of certain technical difficulties. Functional curves demonstrating direct application of sound into the oval window are therefore not available. However, with the tube inserted in the outer acoustic meatus the cochlear function by ossicular conduction can be measured and compared with that by direct application in the round window. The comparison is shown in figure 1.

The next question is whether the cochlea can be stimulated by acoustic energy which does not enter through either window. The question has prompted a heated discussion in the literature on bone conduction. Bezold had maintained that both in air and in bone conduction the cochlea is stimulated by the piston-like movements of the stapes. Other authors stated the belief that in bone conduction the cochlea is stimulated by rhythmic contractions and rarefactions of the otic capsule. Animal experimentation permitted what could be called the "experimentum crucis" in this problem. If Bezold's teaching of exclusive osteotympanic conduction is right, no reflex should be elicited by bone-conducted sounds after extirpation of the stapes. If function is observed, the teaching of osteotympanic bone conduction as the only pathway of bone conduction must be abandoned.

In figure 2, experiments on rabbits are shown in which the stapes has been either destroyed or extirpated.

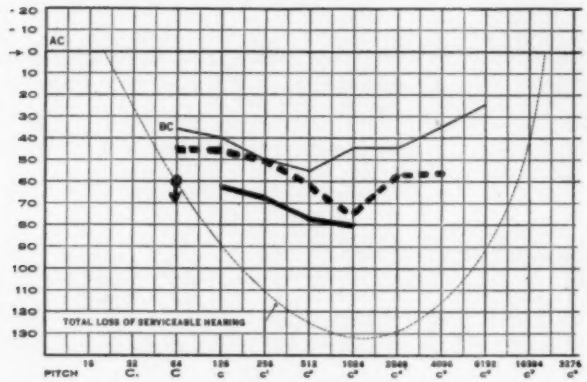

Fig. 1.—Cat; comparison of ossicular sensitivity with aerocochlear sensitivity. The solid line indicates cochlear function by sound conduction through the outer ear. The broken line represents the threshold of hearing by application of sound through the round window.

Fig. 2.—Destruction of the stapes in a rabbit. The footplate is broken. A part of the footplate is pushed into the vestibule. A small hemorrhage is seen in the vestibule. A functional test taken immediately after the destruction of the stapes revealed cochlear function for bone-conducted tones remaining.

The functional tests have to be performed immediately after the operation, because the escape of labyrinthine fluid causes severe cochlear disturbance of its own. After a certain time has elapsed all function is destroyed. Immediately after the extirpation, however, definite function has been observed, showing that *bone conduction is possible without a stapes.*

Fig. 3.—Rabbit; plugging of both cochlear windows with plaster of paris; view of the stapes. Functional tests revealed disappearance of the reflex to both air-conducted and bone-conducted sounds. The loss of a large portion of the plaster of paris is unavoidable in the process of decalcification; hence, only fragments remain.

HEARING IN THE WINDOWLESS COCHLEA

The following experiments aim to show the elastic qualities of the soft tissues in the cochlea. If the soft tissues in the cochlea are easily compressible, undisturbed function for bone conduction is possible even without cochlear windows.

The problem was studied experimentally by transforming the membranous windows into hard, bonelike structures by application of plaster of paris. The histologic findings are given in figures 3 and 4. The functional result has been clearcut.

When one window only was made rigid, bone conduction remained unchanged or even improved (fig. 5). When the regions of both

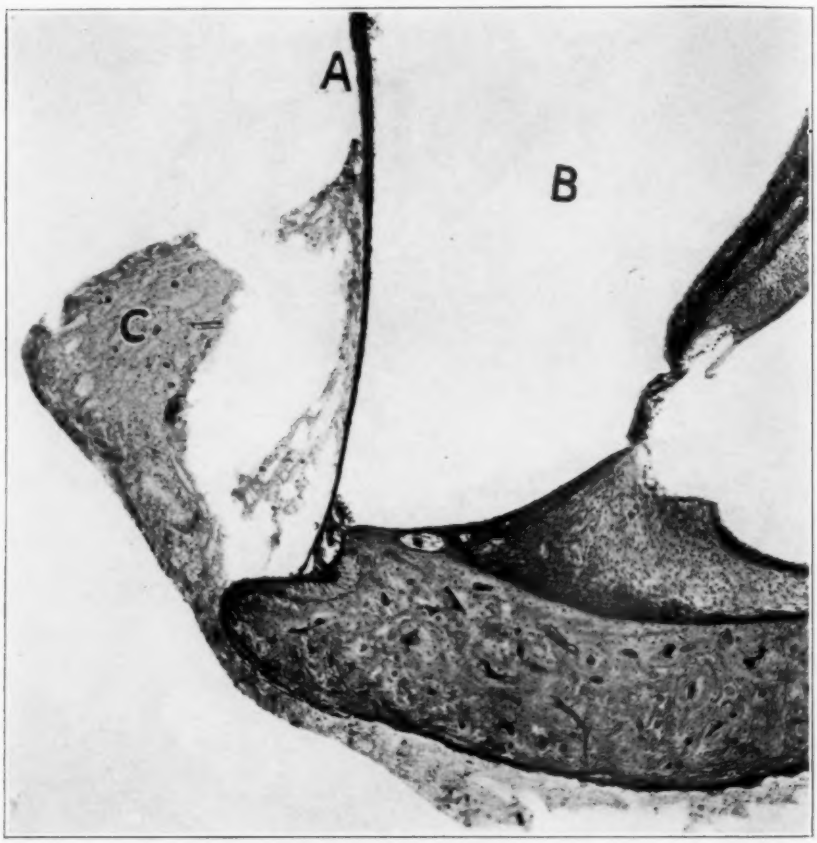

Fig. 4.—Rabbit; plaster of paris in the round window niche. *A*, indicates the round window membrane; *B*, the scala tympani, and *C* the plaster of paris.

windows were made rigid, function could not be demonstrated either by air or by bone conduction.

The experimentation with plaster of paris made a few tests necessary in order to determine the eventual chemical activity of the gypsum. It was to be demonstrated beyond doubt that the plaster of paris deposited in the window niche acted only physically without producing chemical changes in the inner ear. The following experiment seemed to clarify this point. The usual mixture of plaster of paris dries and

hardens in about two or three minutes. The hardening process was watched in the mixing bowl. A mixture of plaster of paris which was softer than usual was introduced into the window niches and the acoustical effect observed. The result was clear. There was no loss of bone conduction as long as the plaster of paris remained soft (over one hour). It is evident, therefore, that the effect of the plaster of paris is physical rather than chemical.

THE FUNCTON OF THE INTRALABYRINTHINE FLUID

E. H. Weber, and later Politzer, stated a belief in direct bone conduction, i. e., in the direct conveyance of acoustic energy to the auditory nerve through the lamina spiralis ossea. In the animal experiment it

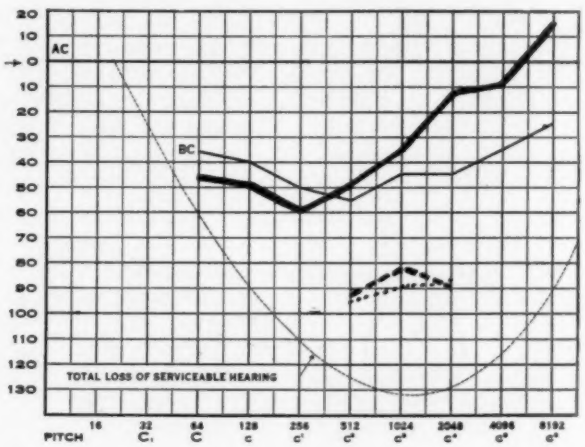

Fig. 5.—Rabbit; the oval window made rigid by plaster of paris. The solid line indicates the normal air conduction curve. The dotted line is the normal bone conduction. The broken line represents the function for bone conduction after the application of plaster of paris.

NOTE: With only one window made rigid, the function for bone conduction is not only unimpaired but even slightly improved.

can be demonstrated that auditory function is possible only in a cochlea in which the labyrinthine fluid is fairly stable. Tapping of the cochlea, resulting in severe loss of endocochlear fluid, destroyed the reflex completely.

Limited lesions in the cochlea, which were permitted to heal, did not disturb the hydrostatic conditions of the cochlea as much as those in the acute experiments. In the case of these healed lesions, function could be demonstrated by means of the reflex.

Direct stimulation of the nerve endings of the acoustic nerve by bone-conducted sound through the lamina spiralis ossea seems to be

impossible. Whatever the pathway is by which the acoustic energy reaches the inner ear, namely, the ossicles, the air of the middle ear or the bone; wherever the sound may enter the cochlea, namely the footplate of the stapes, the round window or the otic capsule, the intralabyrinthine fluid is the last link in the sound conduction system. For this reason it may be called *"the final common path of sound conduction."*

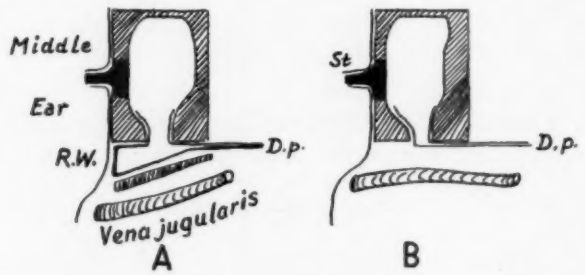

Fig. 6.—Demonstration of the relation between the ductus perilymphaticus and the round window in birds (after Beyer). *B* shows the ductus perilymphaticus (*D. p.*), the only outlet of the inner ear. In *A* the aqueduct has formed a membranous enlargement which reaches the cavity of the middle ear, namely, the round window (*R. W.*).

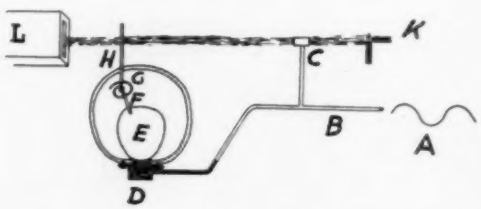

Fig. 7.—Schematic explanation of the method: Sinusoid pressure changes are produced at *A* and led through the tubes (*B*) to both the optical manometer (*C*) and the attachment (*D*). Here the pressure is transferred through a hole in the skull to the cranial cavity (*E*). The attachment (*D*) is put air tight over an opening in the skull. The shaded area in the tube is filled with Ringer's solution. In the cochlea (*G*) is a capillary (*H*), the meniscus of which is optically recorded on the optical kymograph (*L*). The optical system is only shown as the arc lamp (*K*). To be measured are the physical characteristics of the craniolabyrinthine communications (*F*), which anatomically consist of the aquaeductus, the blood and the lymph vessels. Recorded by this method are pressure changes in the cranial cavity and the resulting movements of the intralabyrinthine fluid.

THE CRANIOLABYRINTHINE COMMUNICATIONS

Anatomically the craniolabyrinthine communications consist of the aquaeductus, lymph spaces, the blood vessels and the internal auditory meatus. These anatomic structures cannot be treated individually in functional studies. It is more feasible to consider the group of canals as a functional entity, for which the term "craniolabyrinthine com-

munications" may be used. Comparative anatomy as well as physiologic considerations show that they should be considered a "third cochlear window" (fig. 6).

The physical characteristics of this system of canals have been studied by a method illustrated in figure 7.

As demonstrated in figure 8, slow sinusoid pressure changes in the cranial cavity produce rhythmic movements of the meniscus in the cochlear capillary. Even small pressure changes in the cranial cavity produce movements in the labyrinth.

Fig. 8.—Rabbit; capillary in the basal coil of the cochlea; sinusoid pressure changes in the cranial cavity. The labyrinthine fluid moves according to the pressure changes in the cranial cavity. The phase difference is due to the resistance in the craniolabyrinthine canals. The amplitude of pressure is equal in both experiments. The frequency, however, is increased in experiment *B*. The decrease of the movements of the fluid is due to the increase of frequency. *MC* indicates the meniscus of the capillary in the inner ear, and *P*, the pressure in the cranial cavity.

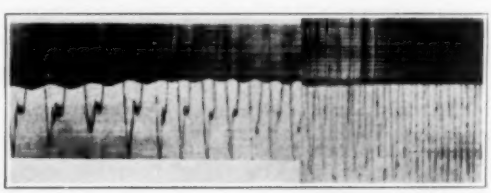

Fig. 9.—Same experiment as in figure 8. The frequency of the pressure changes was increased while the picture was taken. Parallel to the gradual increase of the frequency there is a diminution of the amplitude of the movements of the meniscus. At a certain speed movements of the fluid cannot be recorded; the meniscus appears to be standing still.

A localized increase in pressure in the perilymph without an increase in pressure in the cranial cavity (a condition which in analogy to the eye may be called "glaucoma labyrinthi") is difficult to understand as long as the communications with the cranial cavity are open.

If the frequency of the pressure changes in the cranial cavity is increased (the amplitude of pressure remaining equal) the movements of the labyrinthine fluid are diminished. If the frequency is increased further movements of the fluid cannot be recorded (fig. 9). The

amount of movement of the fluid in the craniolabyrinthine communications shows an important difference between static and dynamic pressure changes. Low pressure changes are easily compensated. For high frequency pressure changes the system shows high resistance to movements of the fluid. In an electrical analogy, the craniolabyrinthine communications act as an inductance coil permitting free flow of direct current but inhibiting alternating current.

This fact may have some bearing on the function of the cochlea, as the following considerations may show:

If the stapes moved with infinite slowness, the difference in pressure between the cochlea and the cranial cavity would be compensated completely by movements of the fluid back and forth through the aqueductus (isotonic conditions in the cochlea).

The opposite extreme is an infinitely fast movement of the stapes. No intralabyrinthine fluid would move through the craniolabyrinthine communications. The vibrations of the stapes would create a maximum of pressure changes (isometric condition in the cochlea).

If it is assumed that isometric conditions in the cochlea are optimal and isotonic conditions least favorable for cochlear function, it is understood why an increase in frequency improves the function of the cochlea. This brings the sensitivity of the ear into the discussion. It is assumed that the causes for the curved shape of the sensitivity curve are manifold. The resonance of the sound conduction system is one important item. Fundamental laws on stimulation of the nerves, as, for example, Nernst's law, must be considered with the other factors. In this paper the physical characteristics of the craniolabyrinthine communications are added as a potential factor.

The left half of the sensitivity curve, (i. e., the lower tone limit to the optimum of the curve) could be partially explained as resulting from a gradual shift from isotonic to increasingly isometric conditions. The optimum is reached at the frequency where isometry is at the highest value. The lower tone limit is the frequency with the minimum of isometry compatible with cochlear function. The right half of the sensitivity curve can be explained by nerve-physiologic considerations.

IMPORTANCE OF PHYSIOLOGIC EXPERIMENTATION

In the past, the anatomic structures of the inner ear have been studied intensively. Each part of the cochlea has been diligently described and carefully measured. The function of each part, however, has not been studied satisfactorily. The students of hearing theories, therefore, have based their assumptions mostly on structural and not on functional considerations. This is unfortunately one sided and may be the reason that several auditory phenomena cannot be explained on the present anatomic basis.

The experiments described in this paper aim to show that certain factors can never be analyzed by anatomic technic but are demonstrable by physiologic methods.

More physiologic experimentation on the function of the structures of the inner ear seems to be a most desirable "Next Step in Auditory Research."

SUMMARY

The value of animal experimentation in the study of the function of the inner ear is discussed.

The acoustic reflex contraction of the intrinsic muscles of the middle ear is a simple, reliable indicator of cochlear function in animals and in man.

The conduction of the sound in the inner ear is studied. It is demonstrated that sound may reach the cochlea through the air of the middle ear, enter it through the round window and be conducted through the scala tympani *("reversed sound conduction in the cochlea")*.

The intralabyrinthine fluid is an essential conductor of acoustic energy. Disturbance of the cochlear fluid is incompatible with good function. Direct conveyance of acoustic energy through the bone to the nerve ending is not possible. Regardless of the portal through which the sound enters the cochlea, the intralabyrinthine fluid is the *final common path* of sound conduction.

Bone-conducted sound can stimulate the cochlea even in the absence of the stapes. Bezold's teaching of osteotympanic bone conduction must be abandoned.

In a windowless cochlea the compressibility of the soft tissues is insufficient for good function.

The resistance of the craniolabyrinthine communications is a function of the frequency of the pressure changes. In the windowless cochlea the system of canals is incapable of acting as a "third cochlear window."

SIMPLE MASTOIDECTOMY

A CRITICAL ANALYSIS OF ONE HUNDRED CONSECUTIVE CASES

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FRAMINGHAM, MASS.

This study was undertaken for the purpose of determining various factors in the preoperative, operative and postoperative stages in the cases considered and evaluating them to aid in the conduct of treatment in similar cases in the future.

For this purpose, I have taken a series of 100 consecutive simple mastoidectomies which were all performed by me in the same hospital during the years 1935 and 1936. The operative technic and postoperative care were the same in all cases.

During the operative procedure, all tissue within the mastoid or connected with it which was infected or might later have become so by extension was exenterated.

To this end, the teachings of Meltzer [1] were found to be extremely useful. The position of the lateral sinus, once determined, gave much information concerning the relative size of groups of cells in various locations. I should emphasize the necessity of complete eradication of the zygomatic, the posterior angle, the parafacial and the paralabyrinthine cells. I also concur in the opinion of Mosher [2]— and follow his practice—that removal of the mastoid tip is not routinely necessary unless it is fractured,[7] necrosed or perforated.

Full exposure of the tegmen and the plate of the lateral sinus was carried out in all cases, with free exposure of the dura of these regions, as advocated by Boies,[3] in doubtful conditions, when the operative findings in the mastoid did not seem sufficient to explain the clinical symptoms. No harmful effect has been observed in any of the cases in which such exposure was carried out.

Much care was taken in closing the wound, by through and through sutures, that there should be approximation of the edges of the periosteum, this being considered to aid in the formation of new bone in the cavity.

Presented as a candidate's thesis to the American Laryngological, Rhinological and Otological Society, Inc., 1939.

1. Meltzer, P. E.: The Mastoid Cells: Their Arrangement in Relation to the Sigmoid Portion of the Lateral Sinus, Arch. Otolaryng. **19**:326 (March) 1934.

2. Mosher, H. P., in discussion on Boies.[3]

Of the 12 cases of recurrent mastoiditis to be reported, in 6 the previous operation had been done by myself with this technic, and in all 6 much new bone was found at the second operation.

Dressings were changed daily. Drains, which had been placed from the antrum through the lower angle of the wound, were loosened every three days and removed when drainage from the middle ear ceased, which occurred after an average of seven and six-tenths days.

The orthodox compression bandages were not used, the dressing consisting of a flannel square with four tapes, tied on the top of the

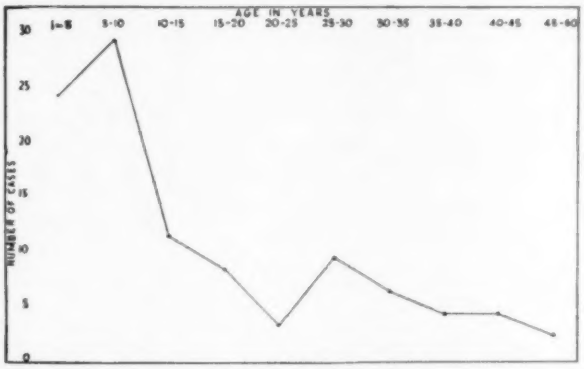

Chart 1.—Age incidence in 100 cases of mastoiditis

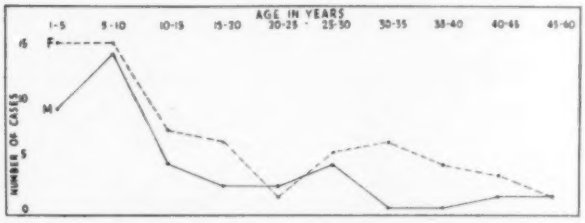

Chart 2.—Sex incidence in 100 cases of mastoiditis.

head. The use of this dressing has resulted in the development of much less edema of the tissues and allowed the saving of much time in applying dressings.

The patients ranged in age from 5 months to 58 years (chart 1); 24 were under 5 years of age and twenty-nine between 5 and 10 years of age, with a very sharp decline in the number in each succeeding five year period. The average age was somewhat younger than that reported by Boies.[3]

There were 67 females and 33 males in this series (chart 2). It is felt that the preponderance of females can be partially accounted for

3. Boies, L. R.: Extradural Inflammation: Study of Its Occurrence in Acute Surgical Mastoiditis, Tr. Am. Acad. Ophth. **41**:150-162, 1936.

by the number of mothers who were thought to have contracted their infection from their children, as later reported.

Little variation was found in the lateralization of infection, the right side having been affected in 50 cases, the left side in 41 and both sides in 9.

Tabulation of the occupations of those affected (table 1) showed the largest group, numbering 39, to be of early school age, 5 to 10 years

TABLE 1.—*Occupation*

School child	39	Radio operator	2	Cabinet maker	1
Preschool child	30	Laborer	2	Painter	1
Housewife	20	Shoe worker	1	Farmer	1
Nurse	2	Mechanic	1		

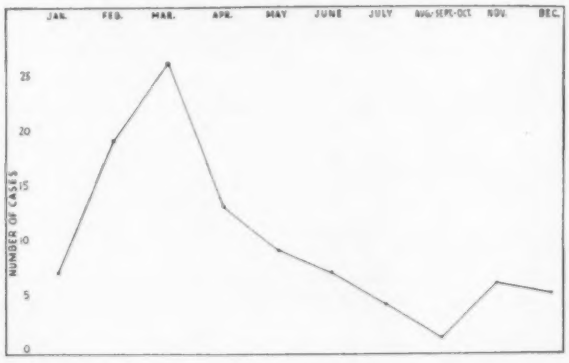

Chart 3.—Seasonal incidence of mastoiditis in the group of 100 cases.

old. The next largest group, numbering 30, was made up of children under 5 years of age. Housewives numbered 20, of whom 18 were mothers of children under 10 years of age who had personally attended their children during attacks of otitis media shortly before. In 3 instances, both mother and child were operated on within a few weeks.

The seasonal incidence, as shown by chart 3, which closely paralleled that reported by Kopetzky and Hadjopoulos,[4] was most significant. The incidence was greatest, 26, in March, dropping sharply to 13 in April, declining fairly rapidly to the low point in August, September and October, then rising slowly to 7 in January and from that point again rising sharply to the highest level.

4. Kopetzky, S. J., and Hadjopoulos, L. G.: Relationship of Upper Respiratory and Alimentary Tract Flora to Mastoid Infections, with Particular Reference to the Epidemiology of Mastoiditis, Laryngoscope **42**:661-673 (Sept.) 1932.

This curve followed closely that of infections of the upper part of the respiratory tract in the community, and such an infection was found to be the immediate preceding illness in 51 of the 74 cases in which a definite history could be obtained (table 2).

Linked with the attacks preceded by infections of the upper part of the respiratory tract might be 5 preceded by acute tonsillitis, 3 by grip, 3 by acute sinusitis and 3 by pneumonia.

There seems to have been a definite etiologic relation between the prevailing disease of the upper part of the respiratory tract and the close association of the younger children with the mothers who attended them when ill, and many infections may have been transmitted from one to another.

TABLE 2.—*Preceding Illness*

Infection of the upper part of the respiratory tract	51
Acute tonsillitis	5
Acute sinusitis	3
Pneumonia	3
Grip	3
Rubeola	7
Rubella	1
Varicella	1

TABLE 3.—*Associated Systemic Condition Present at Operation*

Pregnancy		3
Disease of the respiratory tract		9
Upper part of the respiratory tract	6	
Bronchopneumonia	3	
Acute sinusitis		1
Depressive psychosis		1

ASSOCIATED SYSTEMIC CONDITIONS

A number of associated systemic conditions present in these patients at the time of operation should be commented on (table 3).

1. *Pregnancy.*—One patient was operated on at the three month and 2 at the eight month stage, with no disturbance of pregnancy or of the postoperative course. Ether, without nitrogen monoxide induction, was used as the anesthetic, after consultation with the obstetric attendant.

2. *Disease of the Respiratory Tract.*—Five patients showed no ill effects but in 1 definite bronchopneumonia was found the day after operation. In 3 children operated on during the declining stage of bronchopneumonia, that disease was not exacerbated. Avertin with amylene hydrate was the anesthetic chosen in all cases of involvement of the respiratory tract.

3. *Acute Maxillary Sinusitis.*—One adult with active maxillary sinusitis, likewise operated on under anesthesia induced by avertin with amylene hydrate, suffered no ill effect, the sinus having been opened at the same time.

4. *Depressive Psychosis.*—In a woman of 35 in whom, during many years of chronic purulent otitis media, a depressive psychosis with suicidal tendency had developed, which condition she herself ascribed to the embarrassment of the foul discharge, operation and cessation of the discharge were followed by almost immediate recovery from the depressed state. There has been no recurrence in three years. Here, of course, the cause of the psychosis and the curative effect of operation cannot be positively demonstrated.

TABLE 4.—*Classification of Cases*

1. Cases of acute involvement Those in which a history of previous operation or chronic otitis media was not given	70
2. Cases of chronic involvement Those in which history of chronic purulent otitis media was given; the shortest period in such history was six years; no patient presented a history or diagnostic findings indicating need of radical operation	15
3. Cases of recurrence	12
4. Cases of revision Those in which the patient was referred to my service because of threatening symptoms following a previous operation	3

CLASSIFICATION OF CASES

For the purpose of this study the cases have been divided into four groups (table 4).

Cases of Acute Involvement.—In the first group observations were made on the following subjects: (1) effect of previous attacks of otitis media on the incidence of complicating conditions; (2) effect of paracentesis and of spontaneous rupture on the subsequent course of mastoiditis; (3) optimum time for operation, and (4) virulence of infection in exanthematous disease.

1. Effect of previous attacks of otitis media on the incidence of complicating conditions.

From 18 of the patients a history of previous otitis media with recovery was obtained, but in only 2 of these was any complicating condition observed. One, in whom the attack was preceded by varicella, had a subperiosteal and a perisinal abscess; the other, in whom it was preceded by infection of the upper part of the respiratory tract, had an epidural abscess.

2. Effect of paracentesis and of spontaneous rupture on the subsequent course of mastoiditis.

Paracentesis had been performed on 77 per cent of the tympanic membranes, and 23 per cent had ruptured spontaneously. While I am unable to demonstrate any definite differences in the course of the disease in these two groups, I believe that it is preferable to perform paracentesis to give the patient relief from pain and to promote drainage, since intramastoid pressure, as well as infection, is regarded as a cause of necrosis in mastoiditis.[5]

3. Optimum time for operation (table 5).

Three patients, all under 10 years of age, were operated on during the first week of otitis media, all having shown fulminating infection.

In 1, with operation on the sixth day, a perisinal abscess was found. In this patient pneumonia developed and death occurred on the fourth day. A second patient, with operation on the seventh day, showed a

TABLE 5.—*Incidence of Complicating Lesions*

Time of Operation	Number of Cases	Percentage of Complicating Lesions	
		Preoperative	Postoperative
First week	3	66	33
Second week	19	11	26
Third week	29	7	0
Fourth week and later	19	36	21

tense subperiosteal abscess, with a septic temperature, but recovered promptly without further complication. A third, with operation on the fifth day, because of an apparently septic course, showed no complication and recovered uneventfully.

Nineteen patients were operated on during the second week of otitis media, with complicating conditions appearing preoperatively in 11 per cent and developing after operation in 26 per cent. At operation 2 showed perisinal abscesses; postoperatively, erysipelas developed about the wound in 1 and thrombosis of the sigmoid sinus in another, while in 3 septicemic symptoms developed and blood cultures contained a nonhemolytic streptococcus.

The 29 patients operated on during the third week of otitis media showed the lowest incidence of complicating conditions, 7 per cent preoperatively and none postoperatively. At operation 1 presented mild labyrinthine symptoms and in another an epidural abscess was found.

All of those who were operated on after the third week were considered together, although the time of operation varied from the

5. Kopetzky, S. J.: Otologic Surgery, ed. 2, New York, Paul B. Hoeber, Inc., 1929, pp. 15-18.

twenty-third to the ninety-first day. This group included 19 patients and showed a high incidence of complicating conditions, preoperatively 36 per cent and postoperatively 21 per cent. At operation 3 patients presented labyrinthine symptoms, 2 had subperiosteal abscesses, and 2 had perisinal abscesses; while postoperatively, septicemia developed in 3 and bronchopneumonia in 1.

Too few patients were operated on during the first week of otitis media to permit drawing conclusions from their results, though it seems advisable not to operate so early in the absence of a definite complication.

About the third week is the optimum time for operation, because of coalescence and localization of infection, only conditions advancing rapidly toward complications warranting earlier operation. There is definitely greater danger of complicating lesions occurring, especially preoperatively, as the fourth week and later periods are reached, there being greater likelihood of the infection having reached vital visceral structures.

4. Virulence of infections in exanthematous disease.

Of the 9 attacks preceded by acute exanthematous infection, rubeola preceded 7 and rubella and varicella 1 each. With 1 exception, all the patients came to operation with considerable elevation of temperature; all had extensive destruction in the mastoid area, and in 3 perisinal abscesses were found at operation. The circumstances may be construed to indicate, as shown by Harrell,[6] that the invasiveness and virulence of the hemolytic streptococcus are increased in contagious diseases, this organism having been found in 8 of the 9 patients.

Cases of Chronic Involvement.—The conditions previously classified as chronic were, it must be remembered, so called only because the acute mastoiditis occurred in patients who had had long-standing chronic purulent otitis media.

Contrary to my expectations there were few complicating conditions in this group. Only 2 patients showed preoperative symptoms of labyrinthine irritation; in none did any postoperative complication develop, and the average stay in the hospital was shorter than that with the acute conditions.

The work of Almour[7] leading to his recommendation that all eburnated bone need not be removed in radical mastoidectomies has been noted. While the operations here reported were performed for an acute condition, the same reasoning should apply so far as the eburnated bone is concerned.

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6. Harrell, V.: Hemolytic Streptococcus Mastoiditis, Ann. Otol., Rhin. & Laryng. **46**:194-202 (March) 1937.

7. Almour, R.: The Practical Application of Wittmaack's Theory of Pneumatization, Tr. Am. Acad. Ophth. **37**:315-332, 1932.

In these cases, acute mastoiditis was superimposed on chronic purulent otitis media. Simple mastoidectomy was performed. All eburnated bone was removed because of the difficulty in determining whether there was complete eburnation in a given area and the consequent possibility of isolated islands of infected cells and diploic tissue being present in the midst of the eburnated bone.

The opinion that this procedure should be followed has been voiced by Josephson,[8] and it has been supported by the observation in all my patients that the mastoid was not completely eburnated but that some cells and diploic tissue were present under and within the sclerotic bone.

Twelve of these patients have been completely cured. Three still have some discharge, with no other symptoms, and in these 3 there was probably more extended infection outside the mastoid process, and a radical operation may later be required.

I believe that if all these patients had had radical mastoidectomies without the removal of all eburnated bone with the enclosed infected cells and diploic tissue none of them would have been cured.

TABLE 6.—*Interval During Which Operation Was Repeated*

First six months	2
Second six months	3
Second year	3
Third year	3
Fourth year	1

I am inclined to think, because of these observations, that for many chronic suppurative conditions radical mastoidectomy may be avoided and that operation may safely be limited to simple mastoidectomy. If no infected cells or diploic tissue is found in the mastoid, the operation should be extended to radical mastoidectomy.

Cases of Recurrence.—In the group with recurrent mastoiditis, 12 in number, all patients had had at least one, and some several, previous simple mastoidectomies.

Their ages ranged from 3 years to 28. The interval since previous operation varied from two months to four years (table 6). Practically all attacks were preceded by infection of the upper part of the respiratory tract, with a short period of otitis media, averaging five and one half days, by the end of which time rapid infection and breaking down of the old wound had occurred.

The formation of new bone in these patients was carefully investigated at operation. In no patient whose previous operation had been done within a year could any new bone be demonstrated, but in

8. Josephson, E. M., in discussion on Almour.[7]

every one operated on previous to that period some new bone was formed, with the amount greater in the younger patients.

In all these patients the postoperative course was uneventful, and in no one was there any complicating condition.

Cases of Revision.—In the first patient on whom a revision operation was performed paralysis of the left facial nerve with weakness of the right facial nerve of ten days' duration had developed, following bilateral simple mastoidectomy two months previously. Hearing in both ears had also been lost completely. Records of the previous operation and after-course suggested labyrinthine disease at that time. Complete return of the function of the facial nerves followed within seven days after they were exposed bilaterally in their mastoid course. Much necrotic tissue was found in unremoved parafacial cells.

TABLE 7.—*Complicating Lesion*

A. Present at time of operation	
1. Irritative labyrinthitis	4
2. Perisinal abscess	5
3. Subperiosteal abscess	4
4. Epidural abscess	1
5. Paralabyrinthitis	1
6. Paralysis of the facial nerve	1
B. Developing after operation	
1. Irritative labyrinthitis	1
2. Septicemia	6
3. Bronchopneumonia	2
4. Metastatic abscess	3
5. Suppurative adenitis	2
6. Thrombosis of the sigmoid sinus	1
7. Erysipelas about the wound	1
8. Secondary hemorrhage from the wound	1

The second patient, 41 years old, had had simple mastoidectomy twenty-two days before. Because of persistent facial and orbital pain, a revision operation had been done seven days before, with no improvement in symptoms. Roentgenograms of the petrosa were normal, but at operation a sinus was located beneath the semicircular canals, from which several drops of foul pus was drained. Recovery, while slow and gradual, was complete.

In a third patient, whose wound had failed to heal after a simple mastoidectomy done two and one half years before, a revision operation disclosed infected residual zygomatic cells, and removal of these resulted in complete closure and uneventful recovery.

In all these revision operations it was felt that the cause of the complications mentioned was an incomplete primary operation.

COMPLICATING CONDITIONS

The complications in this series fall naturally into two groups (table 7).

1. *Complications Present at the Time of Operation.*—Of the 4 infections with labyrinthine symptoms, 3 occurred in patients with sclerotic mastoids who had had long-standing chronic purulent otitis media and 1 in a patient with an acute hemorrhagic condition of the mastoid. All vestibular symptoms disappeared within twenty-four to forty-eight hours after simple mastoidectomy. In the patient with the hemorrhagic mastoid secondary hemorrhage from the wound developed three days postoperatively.

The 5 perisinal abscesses were all in children, only 1 of whom had a history of previous otitis media.

Four subperiosteal abscesses occurred in children under 8 years of age.

One epidural abscess was found in a child of 5 months.

2. *Complications Developing After Operation.*—The single case in which irritative labyrinthitis occurred postoperatively was that of a child with a hemorrhagic mastoid, the symptoms being confined to transitory rotary nystagmus on the third postoperative day. In this patient septicemia and a metastatic abscess yielding a nonhemolytic streptococcus on culture also developed.

Although there were others whose symptoms suggested a septicemic condition,[9] only those with a positive blood culture were so classified. Of these there were 6.

COMMENTS

The following subjects will be commented on: (1) anesthesia, (2) organisms present, (3) characteristics of infections, (4) pain, (5) tenderness, (6) sepsis, (7) interpretation of roentgenograms, (8) observations on the blood, (9) mortality and (10) postoperative hearing.

1. *Anesthesia.*—The most satisfactory anesthesia was obtained with nitrogen monoxide and ether, which were used with 46 patients. Ether without nitrogen monoxide induction was used with 21 patients considered too young for satisfactory use of nitrogen monoxide. Avertin with amylene hydrate as a basic anesthetic, with anesthesia sustained by ether, was used with 33 patients, and, while satisfactory anesthesia was obtained, immediate postoperative convalescence was less rapid and satisfactory than in cases in which ether was used.

2. *Organisms Present* (table 8).—Cultures of material from the mastoids showed 54 per cent to contain Streptococcus haemolyticus, 9 per cent Streptococcus nonhaemolyticus and 12 per cent Pneumococcus, while 21 per cent yielded no growth. It was found that some of the

9. Williams, A. W.: Streptococci in Relation to Man in Health and Disease, Baltimore, Williams & Wilkins Company, 1932, pp. 244-246.

culture mediums contained blood serum from a person later shown to be immune to Str. haemolyticus, and so many of the sterile cultures probably should have shown Str. haemolyticus.

3. *Characteristics of Infections.*—In general it may be said that in cases of streptococcic infection there were a more fulminating onset, a more active course of the disease as shown by elevations of temperature and of the leukocyte count and a greater proportion of complicating conditions.

In cases of pneumococcic infection there were a paucity of symptoms, a minimum of reactions of the temperature and blood and a much greater amount of destruction of bone than would have been expected from the symptoms observed.

4. *Pain.*—The absence of pain was not regarded as having negative significance, since many patients with extensive infection and destruction had little or no pain, as in the pneumococcic infections previously mentioned. However, pain, when present, was significant. Pain extending

TABLE 8.—*Organisms in Culture of Material from Mastoid*

Str. haemolyticus	54
Str. nonhaemolyticus	9
Pneumococcus	12
Staph. albus	1
Staph. aureus	1
Diphtheroid bacillus	2
No growth	21

over the mastoid proper and downward into the neck usually indicated an active infection of the tip. In the presence of temporal and parietal pain, operation almost always revealed necrosis of the tegmen or infection of the zygomatic cells. Sudden cessation of pain was noted in several patients in whom subperiosteal abscesses later developed.

5. *Tenderness.*—This was a much less helpful sign than pain, though many infections of the tip were diagnosed by tenderness over the tip and several perisinal abscesses by tenderness over the emissary vein.

6. *Sepsis.*—The absence of septic symptoms did not exclude destruction of bone, but every patient that presented septic symptoms did invariably show disease of the bone.

7. *Interpretation of Roentgenograms.*—While the clinical findings were given the most consideration in deciding whether to operate, roentgen findings were helpful,[10] although in many cases there were discrepancies between the clinical and operative findings and the interpre-

10. MacMillan, A. S.: Personal communication to the author. Schillinger, R.: Roentgen Aspects of Mastoiditis, Am. J. Roentgenol. **39**:192-201 (Feb.) 1938.

tations of roentgenograms. This is not unusual, and, on the whole, interpretations of roentgenograms reached a high standard.[11]

8. *Observations on the Blood.*—Simple elevation of the leukocyte count did not have much significance, and no blood picture distinctive of mastoiditis was found.[12] Repeated differential counts according to the method of Schilling gave much help in prognosis, and the ratio between the total leukocyte count and the percentage of polymorphonuclear cells, especially when tabulated on McKernons' charts, as advocated by Kopetzky,[5] proved of real prognostic value (chart 4).

9. *Mortality.*—In this series 99 patients recovered and 1 died. The latter, a child of 6 who was admitted with a fulminating infection, showed a perisinal abscess at operation, on the sixth day of otitis media. On

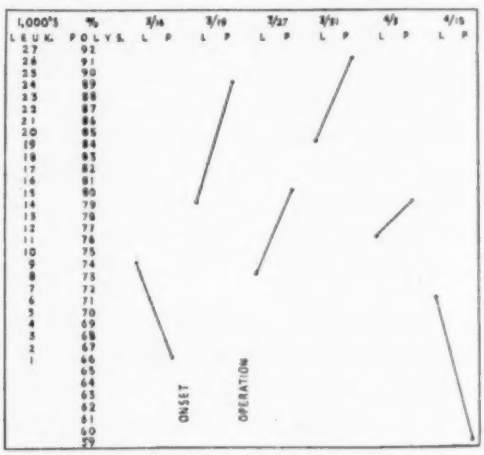

Chart 4.—Resistance chart for a case of sinus thrombosis in which recovery occurred.

the following day definite bronchopneumonia was evident, and death occurred on the fourth day. The pneumonia was regarded as the direct cause of death, but autopsy not having been permitted, factors related to the mastoid infection could not be excluded.

10. *Postoperative Hearing.*—Practically all the patients listed as having chronic infections now have a definite loss of hearing, as might be expected after long-continued chronic purulent otitis media, but in no case is it thought by the patient himself that this loss is greater than that

11. Brownell, D. H., and Hauser, J. J.: The Roentgenogram as an Aid in the Diagnosis of Surgical Mastoiditis, Ann. Otol., Rhin. & Laryng. **47:** 240-246 (March) 1938.

12. Rosenwasser, H., and Rosenthal, N.: The Blood Picture in Otitic Infections, Arch. Otolaryng. **14:**290-308 (Sept.) 1931.

existing before operation. If radical mastoidectomies had been done there probably would have been greater loss of hearing. In all but 3 of these patients discharge has ceased since operation.

Of the patients with acute mastoiditis, 4 are known to have some loss of hearing.

SUMMARY

An analytic report of 100 consecutive simple mastoidectomies, with 1 death, is presented.

Closure of the periosteum aided in the formation of new bone.

The use of noncompression bandages resulted in less postoperative edema of the tissues.

Mastoiditis was most frequent in those between 5 and 10 years of age. Many mothers of such children are believed to have been infected by them.

The frequency of mastoiditis was greatest in February and March and least in August, September and October.

Paracentesis was believed to be advisable.

The optimum time for operation was found to be about the third week of otitis media. Before that there is little localization, while after that, vital visceral structures are more frequently attacked.

In acute mastoiditis superimposed on chronic purulent otitis media, simple mastoidectomy with the removal of all eburnated bone was found to be sufficient.

In recurrent mastoiditis formation of new bone was not found in less than one year after the previous operation.

A revision mastoidectomy was indicated when sepsis with threatening symptoms was present or when faulty healing had occurred.

Cultures of material from the mastoid yielded in 54 per cent of cases a Str. haemolyticus and in 12 per cent a pneumococcus.

Streptococcic infection was associated with fulminating symptoms and numerous complicating lesions, while pneumococcic infection was practically symptomless but caused extensive destruction of bone.

ATELIOSIS OF THE MANDIBULAR ARCH

CRITICAL COMMENT ON GLOSSOPTOSIS, THE SYNDROME OF PIERRE ROBIN

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NEW YORK

Considerable literature has accumulated dealing with observed cases of malformations resulting from arrest of development of the mandibular or the first branchial arch.

Study has been given to these conditions not only as they exist in the human species but also as they occur in the other amniota.

By far the most frequent conditions falling under this classification in man are micrognathia, cleft palate and harelip. These are examples of malformations existing in viable newborn infants.

Among the teratologic examples of the nonviable are cyclopia and agnathia. While the latter are interesting, they do not engage the attention of the clinician.

The condition of micrognathia is rather frequent among sheep and is called by shepherds "hog-jaw." Young lambs so afflicted cannot suckle or graze and are often destroyed for this reason.

As early as 1822 Geoffroy-Saint-Hilaire [1] described the case of a sheep which showed micrognathia, conjunction of the ears, deformity of the palate bone and fusion of the pterygoid processes of the sphenoid. He termed the condition sphenocephalus.

Taruffi [2] in 1891 attempted a classification of all the defects of the lower portion of the face as forms of a single major condition which he termed hypoprosopoaplasia. He then created subclassifications, such as hypomicrognathia (small lower jaw) and hypoagnathia (absence of the lower jaw), unilateral (also Maurice, 1861; Marc, 1892; Buerger, 1903, and others) and bilateral deformities and deformities of medium degree and of high degree.

From the Department of Otolaryngology of Beth Israel Hospital and Jewish Maternity Hospital, service of Dr. Ralph Almour.

1. Geoffroy-Saint-Hilaire, E.: Philosophie Anatomique, Paris, J. B. Baillière, 1822, vol. 2, p. 97; cited by Ballantyne, J. W.: Manual of Antenatal Pathology and Hygiene, New York, William Wood & Company, 1905, vol. 2, p. 423.

2. Taruffi, C.: Storia della teratologia, Bologna, Regia tipografia, 1891, vol. 6, p. 389; cited by Eley and Farber.[11]

A most excellent description of the entire subject was given in 1922-1923 by Gladstone and Wakeley [3] under the title "Defective Development of the Mandibular Arch." They discussed the various possible developmental defects arising from absence or imperfect development of the central region of the craniofacial axis.

In 1929 Robin,[4] of Paris, published a monograph on glossoptosis. Robin was the first to call attention to the clinical problems arising as a result of micrognathia. He, however, placed particular emphasis on the problem created by the ptosed tongue and gave less attention to the developmental defect (micrognathia) which gives rise to glossoptosis. Fischer's [5] contribution on glossoptosis, appearing in the French medical literature in 1939, was based principally on Robin's monograph and referred to the "syndrome of Pierre Robin." Apparently, Robin was the first to describe glossoptosis and associated phenomena as a clinical entity. As recently as 1936 Turner [6] referred briefly to the syndrome in the fourth edition of his textbook.

Robin discussed micrognathia occurring in infants, the developmental anomaly characterized by retrusion of the mandible, which results in facial deformity and poor dental occlusion in later life. In micrognathia, ptosis of the tongue causes mechanical obstruction of the laryngeal portion of the pharynx. Robin stated that glossoptosis is extremely prevalent and that 3 of 4 children have this defect. However, only 1 of 4 adults past the age of 40 is glossoptotic; the other glossoptotic persons die prematurely, in most cases of illnesses which develop as a result of the decreased vital resistance and increased susceptibility to infection secondary to glossoptosis.

Examination and observation of a male infant [7] disclosed, among other developmental defects, the combination of micrognathia and glossoptosis to which Pierre Robin relates the many and varied manifestations of his syndrome. This infant had cleft of the soft and hard palate and the uvula, micrognathia and glossoptosis. At the birth there was normal vertex presentation, but delivery had to be made by the use of forceps. The general condition of the infant was poor, feedings were not taken well and respiration was labored. No cardiac nor pulmonary abnormalities were found on physical examination which could account for the

3. Gladstone, R. J., and Wakeley, C. P. G.: Defective Development of the Mandibular Arch: The Etiology of Arrested Development and an Inquiry into the Question of the Inheritance of Congenital Defects, J. Anat. **57**:149, 1922-1923.

4. Robin, P.: La glossoptose, un grave danger pour nos enfants, Paris, Gaston Doin, 1929.

5. Fischer, H.: La glossoptose: Le syndrome de Pierre Robin, Paris, Vigot Frères, 1932.

6. Turner, A. L., and others: Diseases of the Nose, Throat and Ear, ed. 4, Baltimore, William Wood & Company, 1936.

7. Delivered at the Jewish Maternity Hospital, Jan. 11, 1937.

symptoms. Respiration became more rapid and irregular on January 16. Dyspnea of the obstructive laryngeal type was manifested by the marked retraction of the xyphoid cartilage and supraclavicular and intercostal spaces occurring with each inspiration (fig. 1, *A* and *B*). There were periods of respiratory movements during which no air apparently entered the lung. The free margin of the ptosed tongue filled in the palatine defect. Roentgenologic examination on January 16 indicated possible collapse of the upper and middle lobes of the right lung, and on January 20, central bronchopneumonia of the lower lobe of the left lung. The temperature rose to 103 F. Respiration was improved by elevating the lower jaw, and tubal feedings were successfully administered for a short time. Later, respiration again became rapid and labored; the temperature fluctuated between 99 and 104 F., and the infant died.[8] Permission for autopsy was refused.

Fig. 1.—Photographs of the patient reported on. *A*, marked retraction of the xiphoid process characteristic of obstructive laryngeal dyspnea. *B*, micrognathism; retraction of the xiphoid process and the intercostal spaces is well marked.

MECHANISM

In this case there were two developmental defects, micrognathism and cleft palate, both manifestations of ateliosis of the mandibular arch. In this circumstance the malposition of the symphysis menti relaxes its attached ligaments, which results in backward displacement of the tongue, obstructing the pharynx and interfering with the free flow of air during respiration.

A variety of theories have been advanced to explain the mechanism of development of micrognathia. Normally, the developing mandibular arch

8. Jan. 27, 1937.

fuses in the midline, forming the symphysis menti. The ventral portion becomes the mandible and the dorsal portion the maxillary process, from which two shelves grow medially, fusing and forming the hard palate and the floor of the nose. Keibel and Mall [9] attributed micrognathia to simple arrest in this developmental process. They called attention to the fact that the fetal upper lip at the fifth month projects much further forward than the lower lip and suggested that an inhibitory process at a later period prevents outward growth of the lower portion which would overcome the disparity in projection between the upper and lower portions. Davis and Dunn [10] called attention to the fact that during uterine life the entire weight of the fetus rests on the vertex of the cranium and that during the same period the sternal region transmits a distinct pressure on the mandible. Since the latter develops and unites in the midline before the maxilla, pressure of sufficient magnitude could conceivably force the partly calcified mandible backward against the softer maxilla, with consequent production of deformity. The concomitant cleft palate in the case here reported, in the 4 cases reported by Eley and Farber,[11] in 2 of 3 cases reported by Lannelongue and Ménard [12] and in the case of Davis and Dunn lend weight to this theory. It is quite conceivable that sufficient pressure to retard the development of the mandible would likewise be able to prevent the fusion of the palate in the midline.

The development of glossoptosis is more readily explained than that of its antecedent, micrognathia. The genioglossi muscles are the principal means of drawing the tongue forward. Their insertion is at the symphysis menti, from which point traction on the tongue is maintained. In micrognathia, the backward displacement of the symphysis renders traction sufficient to prevent the tongue from dropping back an impossibility.

CAUSATION

Glossoptosis may be hereditary, congenital or acquired, the last being the most common according to Ivy.[13] The hereditary deformity can be

9. Keibel, F. K. J., and Mall, F. P.: Manual of Human Embryology, Philadelphia, J. B. Lippincott Company, 1910.

10. Davis, A. D., and Dunn, R.: Micrognathia: A Suggested Treatment for Correction in Early Infancy, Am. J. Dis. Child. **45**:799 (April) 1933.

11. Eley, R. C., and Farber, S.: Hyperplasia of the Mandible (Micrognathy) as a Cause of Cyanotic Attacks in the Newly Born: Report of Four Cases, Am. J. Dis. Child. **39**:1167 (June) 1930.

12. Lannelongue, O. M., and Ménard, V.: Affections congénitales, Paris, Asselin & Houzeau, 1891, vol. 1, p. 423; cited by Eley and Farber.[11]

13. Ivy, R. H.: Congenital and Acquired Defects and Deformities of Face and Jaws: Review of the Literature for 1936, Internat. Abstr. Surg. **64**:433, 1937; in Surg., Gynec. & Obst., May 1937.

traced through several generations. La Page [14] called attention to a case of this type in which the family history indicated that the previous deaths of 3 children were directly related to micrognathia with glossoptosis. Pfaff [15] noted hereditary transmission in 50 cases and traced the defect through several generations in some cases. On the other hand, Gladstone and Wakeley [8] in a study of 554 cases of harelip and cleft palate found the same condition in both parent and child in only 2 cases (0.35 per cent). They stated the belief that the condition is due to malnutrition of the embryo. The most frequent cause of congenital glossoptosis, according to Robin, is syphilis, because of the Treponema pallidum's predilection for developing bone tissue. He gave no scientific evidence to corroborate this assertion. It is questionable whether, as a matter of fact, syphilis is a factor at all. Eley and Farber,[11] who observed 4 cases, did not mention the existence of syphilis in any of the infants, nor was there any evidence of the disease in their case in which autopsy was performed. Acquired glossoptosis may be caused by syphilis, by faulty location of teeth or, most commonly (Robin), by an improper position of the infant while nursing.

In his monograph, Robin traced the course of his syndrome from the development in the growing fetus to the end results in the mature person. The arrest in development of the mandible is regarded as the first step in a series of physiologic and anatomic disturbances which result in secondary somatic pathopoiesis of the entire organism, although all manifestations of the syndrome may not appear in every case. The severity of the symptoms is determined by the degree of glossoptosis, which in turn depends on how pronounced the micrognathism is.

Robin described a space, which he called the "confluent vital fonctionelle" (fig. 2), that includes all the upper respiratory-digestive passages, extending from the basilar vault to the glottis. The bony boundaries of this space are the jaw, the bones of the face, the cranium and the cervical spine. It therefore includes from above downward the nasal cavities, the nasopharynx, the oral pharynx and the laryngoesophageal juncture. Robin abbreviated the term "confluent vital fonctionelle" to C. V. F., an abbreviation which will be used subsequently in this report, as it eliminates the difficulties of finding a suitable English equivalent.

The C. V. F. is not a fixed space but one the form and capacity of which change with alterations in the position of the bony structures comprising the boundaries. In man, the form and capacity of the

14. La Page, C. P.: Micrognathia in the New-Born, Lancet **1**:323, 1937.

15. Pfaff, cited by Brüning, H., and Schwalbe, E.: Handbuch der allgemeinen Pathologie und der pathologischen Anatomie des Kindesalters, Wiesbaden, J. F. Bergmann, 1913, vol. 2, p. 442.

16. Footnote deleted.

C. V. F. are particularly prone to change, as the upright position places the various structures within and bordering it in the relation of reciprocal support. Therefore, in man, a change in the position of but one of these structures may affect the positions of the other structures, eventuating in a profound disturbance of the C. V. F.

Robin developed the concept of his syndrome on this basis. He maintained that the backward displacement of the tongue in micrognathia obstructs air and food passing through the channels comprising the C. V. F., and, in addition, exerts pressure on the articulations, the nerves and the blood and lymph vessels in this region. Normal development and functioning of both contiguous and remote tissues is consequently impeded.

If the ptosed tongue is capable of exerting such pressure, his view may be accepted, for if arterial, venous and lymphatic circulation were impeded and the sympathetic and the last four cranial nerves, which

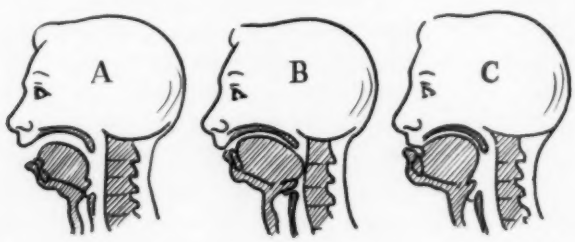

Fig. 2.—The "confluent vital fonctionelle" in the normal subject; position of the mandible and tongue during respiration (*A*) with the mouth open, (B) with the mouth closed and (*C*) with the jaw projected.

traverse this area, compressed, profound trophic disturbances and dysmorphism of the organism would be produced (fig. 3). The syllogism is far reaching if the premise is accepted.

DIAGNOSIS

Congenital or acquired glossoptosis is diagnosed in the following manner: The head of the infant, in the prone position, is held firmly by an assistant, and the examiner separates the lips with the fingers of one hand while slowly and gently pressing the chin backward with the other hand. If there is glossoptosis the lower gingival ridge recedes and is more or less set apart from the upper ridge. The degree of recession is in direct proportion to the severity of the deformity.

PROGNOSIS

The prognosis in glossoptosis depends on the cause of the micrognathism, the degree of mandibular recession, the age at which active treatment is instituted and the type of therapy employed.

The infant usually does not survive if the congenital mandibular deformity produces recession from the maxilla of 10 to 12 mm. or more (Robin). In later life, the micrognathic infant may develop compensatory prognathism as, after eruption of the temporary teeth, he learns that respiration is facilitated by locking the lower over the upper incisors. This prognathism should not be confused with the type seen in *macrognathia.*

Micrognathism is generally more conspicuous in patients in whom the condition had its onset during intrauterine life or before the age of

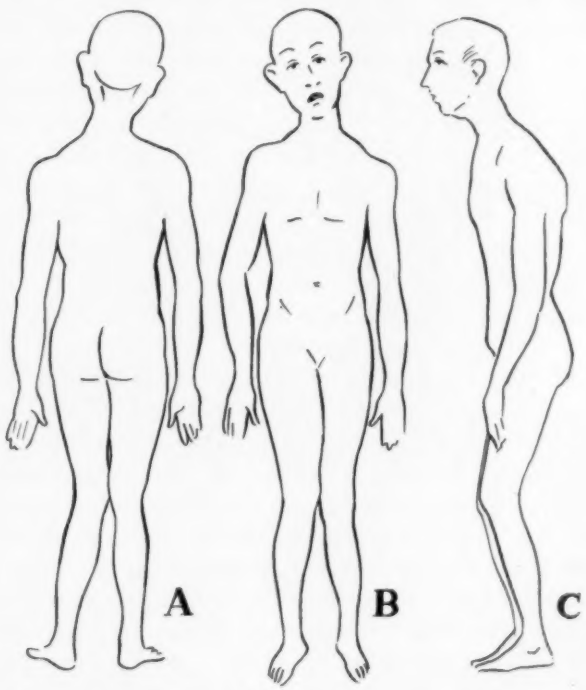

Fig. 3 (after Robin [4]).—Systemic manifestations of glossoptosis: (*A*) from the posterior aspect, protruding ears, kyphosis, scoliosis, lordosis, hands in pronation and genu valgum; (*B*) from the anterior aspect, strabismus, adenoids, carious teeth, erythrodermia, pigeon breast and flat feet, and (*C*) from the lateral aspect, dolichocephaly, harelip, rachitic rosary, abdominal ptosis, cryptorchidism and flexed knees. Glossoptotic subjects are slow in learning to talk and walk, and their mental development is retarded. They are subject to nightmares, tics, convulsions, chorea, headaches, meningeal affections, aprosexia, hyperhidrosis, sensitivity to cold, acrocyanosis, chilblains, varices, deviation of the nasal septum, sinusitis, pulmonary disease, mongolism, disorders of the thyroid and pituitary glands, aerophagy, dyspepsia, gastroenteritis, appendicitis, constipation and enuresis. Senile changes develop more rapidly than in the normal subject.

2 years. If the condition is not treated, all structures bounding the C. V. F. are maldeveloped, and there is consequent malnutrition and inadequate ventilation. According to Ullrich,[17] congenital glossoptosis is more serious than acquired glossoptosis. The postmortem studies in La Page's case indicated that cardiac dilatation was the principal cause of death.

TREATMENT

The treatment of micrognathia has been thoroughly discussed in the medical literature, especially the surgical treatment. Babcock [18] described a method by which the temporomandibular structure in front of the external auditory canal is disarticulated, the mandibular heads forced anteriorly and the gaps filled with costal cartilage. Eley and Farber [11] used a splint of their own devising to hold the jaw forward and reported successful results from this treatment. Hensel [19] performed osteotomy of the ascending rami and grafted bone into the symphysis menti.

The objective in all corrective measures for glossoptosis itself is obtaining a favorable position of the head and jaw. When the head is bent forward, the backward movement of the jaw tends to contract the C. V. F.; conversely, when the head is extended, the C. V. F. is expanded and respiration facilitated (fig. 4). Consequently, the position of the jaw is of fundamental importance.

In advanced stages, suturing the tongue to the lower lip seems to be the indicated measure. However, the danger of infection with the ever present oral bacteria renders the measure objectionable, and it is almost invariably followed by bronchopneumonia. La Page found that the infant nursed and breathed better when in a position approaching complete ventral decubitus with the neck extended; he used the Eley-Farber splints with unsatisfactory results. Mouth breathing is a feature of glossoptotic persons and frequently leads to tonsillectomy and adenoidectomy. Unless the condition of the tonsils and adenoids indicates that they should be removed, there is no more reason for their surgical removal than there is in the normal person. In severe cases tracheotomy may be required.

Davis and Dunn, noting that the young of lower animals reach outward and upward to nurse, stated the belief that glossoptotic infants should be encouraged to nurse in a similar manner. In the case of bottle-fed infants an appliance can be devised so that they stretch their necks while feeding.

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17. Ullrich, G.: Glossoptosis bei Mikrognathie, Deutsche med. Wchnschr. **61**: 1033, 1935.

18. Babcock, W.: The Surgical Treatment of Certain Deformities of the Jaw Associated with Malocclusion of the Teeth, J. A. M. A. **53**:833 (Sept. 11) 1909.

19. Hensel, G. C.: The Surgical Correction of Mandibular Protraction, Retraction and Fractures of Ascending Rami, Surgery **2**:92, 1937.

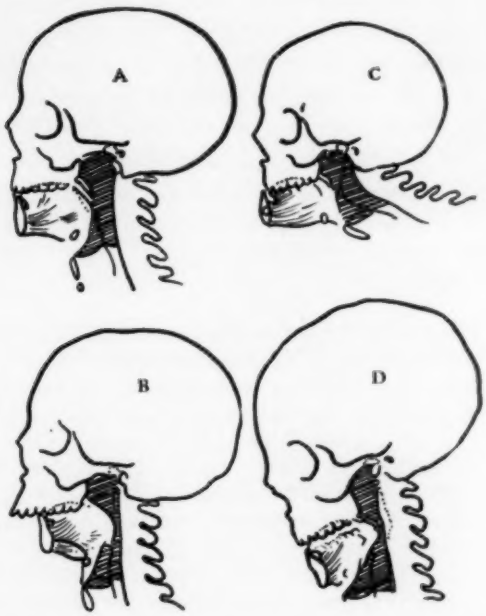

Fig. 4 (after Robin [4]).—Variations in the capacity of the "confluent vital fonctionelle": (*A*) volume in the normal subject; (*B*) decreased volume in glossoptosis with the mouth closed, the posterior position of the mandible dropping the tongue toward the spinal column; (*C*) volume increased in glossoptosis by extension of the head; (*D*) volume decreased in glossoptosis by flexion of the head.

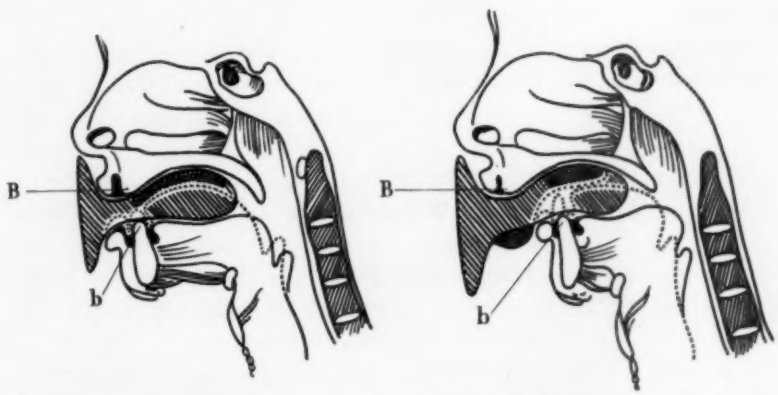

Fig. 5 (modified from Robin [4]).—Left, the mechanical factor: the nipple emptied by compression between *B* and *b*. Right, nursing with mandibular atresia; note the incomplete compression of the nipple due to functional and anatomic changes in the tongue and disturbance in the relation between *B* and *b*. Hatching indicates the nipple full of milk; crosshatching, the nipple emptied.

Robin endorsed this type of feeding, which he referred to as orthostatic nursing, and recommended in addition the divided feeding, i. e., interruption of nursing after six swallows for the purpose of rest. He stated that with breast-fed infants, the weight of the breast rests heavily on the infant's chin during nursing and may thus cause slight recession of the mandible, which becomes progressively more pronounced, with glossoptosis as an ultimate result. Robin stated the belief that an improper position of the infant while nursing is the most common cause of acquired glossoptosis and leads to the same difficulties as are caused by congenital glossoptosis (fig. 5).

According to Robin,

> Decrease in the lower maxillary space results primarily in glossoptosis, particularly in a dropping backward of the floor of the mouth and the base of the tongue, the impingement of which against the spinal column closes the epiglottis, thus bringing about obstruction of the lower part of the respiratory tract and compressing the neurovascular clusters of the functional vital confluent. So, on the one hand, glossoptotic persons are forced to breathe through their mouths, and, on the other hand, they are subject to nutritional disturbances. Not only do they experience difficulties of mastication, digestion and respiration, but the compressions and ptoses bring about frequent and serious disturbance of function in all the vital organs controlled by the sympathetic and central nervous system, as well as skeletal deformities. These considerations clearly indicate the pathogenic importance of glossoptosis and the necessity of treating it as soon as it appears.

COMMENT

In a short critical review, it would be impossible to discuss all points elaborated by Robin. A brief discussion of the more outstanding facts and theories, however, suffices to demonstrate that the disorders that may follow glossoptosis due to micrognathia, for which there are relatively simple anatomic and physiologic explanations, have been expanded, without scientific basis, into a complicated, intricate, all-inclusive syndrome by resorting to gross hypothesizing, which is stated in terms of absolute fact.

It hardly seems likely that the many and varied morbid changes described by Robin could have their origin in glossoptosis caused by micrognathia. In the majority of cases of glossoptosis, the pressure on pharyngeal structures or the impediment to the free passage of food and air is scarcely sufficient to bring about changes which affect the development of the entire organism so profoundly.

A review of Robin's monograph leads to doubt concerning the various morbid changes which he ascribes to glossoptosis. Moreover, it may well be questioned whether such changes could all be caused by micrognathia, and it seems more plausible that the existence of such a large number of marked manifestations can only be based on a more profound genetic anomaly.

The existence of some underlying genetic fault seems plausible from observed coexisting developmental errors. Besides the frequently associated cleft palate, the following conditions are observed: ankylosis of the temporomandibular joints (Taruffi), ankylosis of other joints (Perthes) and congenital clubfoot, scoliosis and muscular weakness (Lannelongue and Ménard).

In this connection Gladstone and Wakeley [8] concluded:

> We believe that the defects in growth of the face and lower jaw are usually associated etiologically with defective development of the brain; for instance cyclops with defective growth of the fore-brain; otocephaly and agnathia with maldevelopment of the hind-brain.

If this conclusion is accepted, a partial explanation, at least, is available for some of the many phases of the syndrome expounded by Robin.

SUMMARY

An infant with congenital micrognathia, glossoptosis, cleft palate and cleft uvula is described and the condition discussed, with a review of the literature and a critical appraisal of the "Pierre Robin syndrome."

Wickersham Professional Building, 133 East Fifty-Eighth Street.

BORDERLINE ALLERGY

ITS RELATION TO HYPERPLASTIC DISEASE OF THE RESPIRATORY TRACT

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AND

SAMUEL PELUSE, M.D.

CHICAGO

The difficulties in properly evaluating the symptoms and pathologic picture of hyperplastic disease of the respiratory tract have resulted in a regimentation which falsely groups the borderline cases in classifications which belie their allergic nature. Physicians have been taught to recognize allergy only by the classic symptoms of obstruction and watery discharge and by the pathologic findings of eosinophilia, edema or extreme grades of hyperplasia. They have entirely forgotten that in the nose, as everywhere else in the body, different symptom complexes exist which are dependent on the degree of pathologic change or the constitutional makeup of the individual at a particular time. Rackemann [1] aptly voiced this in the following comment:

> . . . There are many gradations between those not sensitive at all, and those extremely sensitive. In the middle ground, there are people who are slightly sensitive to one thing or, usually, . . . to a variety of things. I think we shall get farther if we regard the allergic group as simply a variation of the normal group.

As stated by Vaughan,[2] the difference between the allergic and the nonallergic is not so much qualitative as quantitative, i. e., a pathologic exaggeration of a normal physiologic response, and the same assertion holds for manifest and for borderline allergic patients. The latter are those who do not present the classic symptoms and pathologic picture of manifest allergy but present positive reactions to concentrated protein allergens and have or have had other mild allergic manifestations or come from allergic families.

Vaughan coined the term "minor allergy" [2] for such conditions as migraine, eczema and hyperesthetic rhinitis. Of a community of 508

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1. Rackemann, F. M., in discussion on Vaughan,[2] p. 211.

2. Vaughan, W. T.: Minor Allergy: Its Distribution, Clinical Aspects and Significance, J. Allergy **5**:184, 1934.

persons, he found that about 60 per cent suffered from minor or major allergy. This figure is generally accepted as applicable in most communities. However, if a careful study over years is made of the respiratory difficulties of so-called nonallergic members of the families involved, it will be noted that they are chronic sufferers from colds, intermittent nasal obstruction, pharyngitis, recurrent laryngitis and conduction deafness. It will be found further that a great percentage of them become manifestly allergic at some time. Neither of Vaughan's terms suffices to describe the clinical or pathologic picture which they present, and so we propose that they be classified as borderline allergic patients, since their respiratory mucosa definitely reveals pathologic changes.

Careful observation of a large group of such patients over fifteen years has convinced us that those who initially presented symptoms and findings of so-called intumescent, hypertrophic or hyperplastic rhinosinusitis, chronic lymphoid pharyngitis, recurrent laryngitis, pachylaryngitis, nodes and some types of chronic tubal tympanitis become at some time frankly allergic. The difficulties in properly classifying them have been due to the false premise that allergic patients must present the classic symptoms of allergy. The heavy pollen season of 1939 in the Middle West has particularly convinced us of the truth of our early observations. In over 200 patients treated by us for years for hyperplastic conditions of the nose, throat and ears or for frequent colds and nasal suppuration, classic symptoms of hay fever have for the first time developed.

A great deal has been written [3] concerning the character of the formed elements of the mucosal discharge in allergic patients, particularly the eosinophils and the polymorphonuclear leukocytes, as a means of differentiating the allergic from the nonallergic patients. Admittedly about 90 per cent of the manifest allergic patients reveal eosinophilia, but this finding is nonexistent in most patients during the formative period of the allergic condition (fig. 1). The predominating cells are the lymphocytes, plasma cells, histiocytes, polymorphonuclear leukocytes and other wandering macrophages. We therefore feel that the picture of the mucosal discharge is wanting as a test for the differentiation of borderline allergy.

The color and texture of the nasal, pharyngeal or laryngeal tissues are of little diagnostic significance, except in areas exposed to the air current

3. Hansel, F. K.: Allergy of the Nose and Paranasal Sinuses, St. Louis, C. V. Mosby Company, 1936. Cowie, D. M., and Jimenez, B.: Cytologic Examination of Nasal Smears of Sensitized and Nonsensitized Persons with Nasal Symptoms, with Special Reference to Eosinophil Counts and to Simultaneous Blood Counts, Arch. Int. Med. **57**:85 (Jan.) 1936.

or to the drip of the exudates. Ordinarily some hyperplasia is noted throughout the mucous membrane but especially along the anterior tip and pendulous portions of the middle turbinate, where the tissues appear rather transparent, along the lateral pharyngeal walls, where a bar

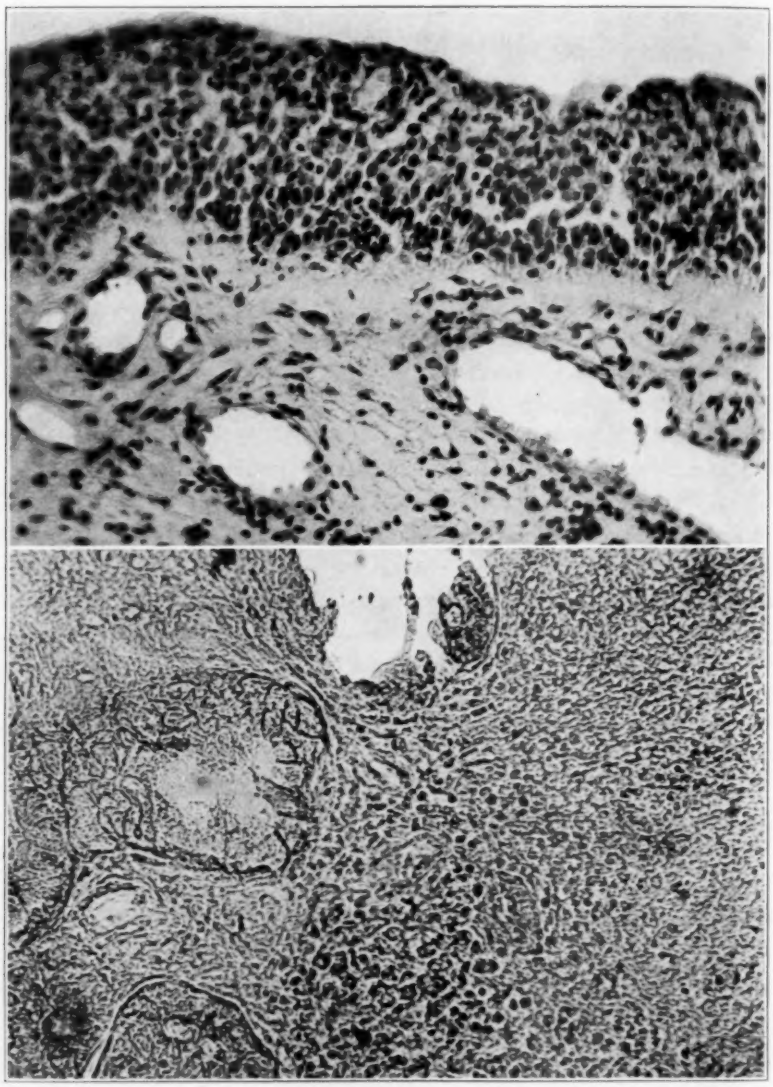

Fig. 1.—Above, tissue from a patient with borderline allergic rhinitis, revealing epithelial hyperplasia, thickening of the basement membrane, numerical increase in all cellular elements, increase in glandular activity and a normal number of eosinophils (hematoxylin and eosin stain; × 246). Below, section of same tissue as shown above but stained by the oxydase technic for the identification of leukocytes, including eosinophils. Note the absence of this group of cells (× 492).

formation is noted, and on the vocal cords, where definite discrete or diffuse thickening takes place. Early in the course of borderline allergy, even these findings may be absent.

The gross pathologic changes which occur in the respiratory mucosa are primarily the result of edema and fibrosis. Between attacks, however, the mucosa may appear normal. The degree of pallor depends on the fluid content of the tissues. It is fair to say that when edema and pallor exist an active allergen is responsible and is coming in contact with the tissues in amounts which are able to cause these changes. It must be remembered, however, that small amounts of allergen are able to cause a gradual thickening of these tissues without an intervening period of acute edema. Also, the color of the tissues in this instance is that associated with hyperemia. The changes correspond with the results of cytologic examination, i. e., during an acute attack (fig. 2), there are pallor, edema and eosinophilia, but in low grade, minor involvement (fig. 1), hyperemia and thickening, with only a few or a normal number of eosinophils in the smear.

Histologically, the tissues reveal the presence of a hyperplastic process (fig. 1). All connective tissue elements are increased, but the classic edema of the tunica propria is missing except to a slight degree. The basement membrane in such cases is frequently greatly thickened, out of proportion to the changes noted in other areas. The cellular elements are preponderantly round cells, histiocytes, fibroblasts, plasma cells and neutrophilic leukocytes. Though eosinophils are present, they occur only in normal numbers. Such elements as subepithelial abscesses and cysts are missing. This type of tissue frequently shows changes in bone, tending to make for a more compact bony structure. Numerous osteoblasts and osteoclasts are noted along the bony surfaces, where new bone can be seen. The blood vessels are much increased in number and show a high degree of periarteritis. The glandular elements appear about normal in number but actively engorged, with a greater preponderance of mucous glands. The tissues reveal without exception occasional organisms usually present in the respiratory flora, such as pneumococci, Friedländer's bacillus, Micrococcus catarrhalis, streptococci, Bacillus influenzae and staphylococci. In such patients a study of the exudates, particularly of nasal smears, gives little information on the allergic character of the condition.

The diagnosis of borderline allergy is made by the finding of gross hyperplastic changes in a person with a history of allergy or in whose family there is a history of allergy. In addition to manifest symptoms, one must be suspicious of a history of frequent colds, intermittent nasal obstruction with postnasal drip, chronic lymphoid pharyngitis, pachy-

laryngitis, singers' nodes and papillomas, chronic purulent sinusitis which does not yield readily to routine treatment and tubal tympanic deafness. The same symptom complexes must be carefully looked for in the immediate family of the suspected borderline allergic patient.

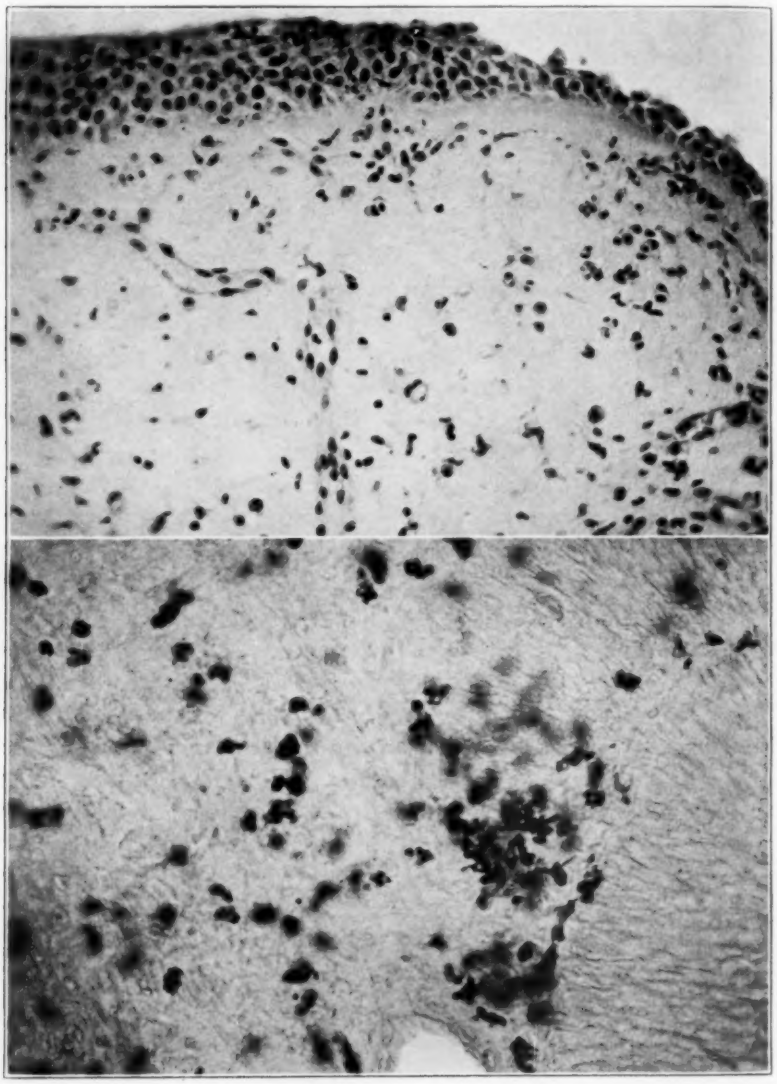

Fig. 2.—Above, tissue from a patient with active allergic rhinitis (hay fever). The appearance is similar to that in figure 1, except for the increase in edema in the substantia propria and the presence of numerous eosinophils (hematoxylin and eosin stain; × 246). Below, section of same tissue as above but stained by the oxydase technic for the identification of leukocytes. Note the great number of these cellular elements (× 492).

Because of the little help ordinarily afforded by precise laboratory data, the history becomes all important in making a diagnosis. A typical family history demonstrating this fact is as follows:

H. G., aged 45, has been treated for years for "chronic catarrhal conjunctivitis." We have seen him numerous times in the past fifteen years because of frequent colds, each followed by residual pansinusitis lasting three to four weeks. The nasal tissues were thickened and hyperemic even between colds, and a mucoid secretion was present more or less constantly in the nasopharynx. On many occasions we also treated both his children for numerous colds. In fact, the children definitely presented a pediatric problem because of this condition. The patient is unable to remember much about his parents, since they died young, and he has no other living relatives except one brother, who is apparently normal. At the age of 8, i. e., six years ago, severe autumnal hay fever developed in his son, which, however, disappeared at the age of 13. He has been free of hay fever for two years. In the daughter, who is two years younger, i. e., 12, severe autumnal hay fever developed during the present (1939) season. Though for the past ten years we have suspected an allergic taint in the parent, the repeated lack of laboratory evidence of eosinophilia, either in the blood or in smears, together with the absence of a family history of manifest allergy, had made us hesitant about classifying this condition as allergic. The appearance of hay fever in the elder child six years ago, however, clarified the situation, so that we were able to complete an allergic survey and more intelligently treat the entire family.

It is not our purpose to present the condition as a new disease, since obviously the borderline type differs from the manifest type only in the degree of severity of the condition at the time of examination. We must realize that the symptoms and findings are merely those which occur in one condition, i. e., hyperplastic disease of the respiratory tract, but for the sake of clarity we shall discuss each as a separate entity. The symptom complexes which we suggest be recognized as possibly allergic are as follows: (*a*) chronic intermittent nasal obstruction with postnasal drip; (*b*) chronic lymphoid pharyngitis; (*c*) frequent colds; (*d*) recurrent acute laryngitis, pachydermia laryngis, singers' nodes and papillomas; (*e*) chronic purulent discharge from the nose in children, and (*f*) tubal-tympanic catarrh.

SYMPTOM COMPLEXES

CHRONIC INTERMITTENT NASAL OBSTRUCTION WITH POSTNASAL DRIP

Chronic, intermittent nasal obstruction with postnasal drip is the result of thickening and irritation of the nasal mucosa. In such cases, intermittent obstruction is present on one side of the nose at a time, changing with the position of the head and affected by weather, alcohol, emotions and irritating vapors but particularly by pollens and other protein dusts. Many patients suffer from headaches, and the majority have associated endocrine hypofunction. Concurrent with these symptoms is an annoying postnasal drip and an accumulation,

particularly on arising, this being the night's secretion from the sinuses. This may be the symptom that brings the patient to the doctor. Short attacks of sneezing may be present, especially on arising, but not the persistent sneezing of hyperesthetic patients. It must be differentiated from purulent sinusitis, though a purulent infection may also be present, particularly in children. In such cases, the nasal smear reveals nothing unusual. Lavage of the nose or sinuses reveals a perfectly clear discharge, with a p_H close to 7.0 and high in protein, so that it appears exceedingly foamy on being shaken in a test tube. The cases described in many textbooks as those of intumescent rhinitis or hypertrophic rhinitis belong to this group. These conditions in time may become severe enough to present the classic symptoms of hyperesthetic rhinitis. It is our feeling that the difference is merely one of degree, the earlier stage being a quiescent state in a person with an allergic tendency, ready when stimulated by the proper allergens to develop into the extreme state of vasomotor imbalance and hyperesthesia manifested in cases of frank allergic rhinitis. It must be remembered that allergic conditions are exudative and that when even a mild allergy exists some exudation occurs. It is this type of patient, so common in rhinologic practice, that has during the present pollen season turned up with frank symptoms and signs of hay fever. This is the type of patient also who frequently suffers from virus colds, chronic lymphoid pharyngitis, laryngeal conditions and tubal tympanic catarrh. Pathologically such patients present grossly and histologically various degrees of hyperplasia and represent the uppermost bounds of a general hyperplastic change noted throughout the respiratory mucosa.

CHRONIC LYMPHOID PHARYNGITIS

Chronic lymphoid pharyngitis is a natural sequence to the condition just described. In the adult, it is characterized by a pharyngeal cough made worse by smoking, a postnasal drip with morning accumulations which have to be coughed up and at times pharyngeal pain due to the bathing of the tissues in secretions all night, which tends to disappear as the day wears on. This group of symptoms is more frequently complained of by adult patients than any other nasal or pharyngeal symptom complex, and yet it is one for which probably the least is done. Obviously, the postnasal drip is due to either a purulent or a nonpurulent process in the posterior sinal group of cells, which may be differentiated by washing out the sinuses and examining the washings. When they fail to reveal pus, one should suspect an allergic condition, particularly when the tissues in the area show hyperplastic changes. Grossly, examination of the pharynx and postnasal area in such patients reveals hypertrophy of the posterior tips of the lower turbinates, the so-called "mulberry type" of turbinate, with postnasal atrophy of the mucosa of the lateral borders of the posterior choanae

and the mucosa of the rostrum of the sphenoid sinus. There is usually decided hyperplasia of the lateral pharyngeal bar and occasionally some hyperplasia of the mucosa of the posterior wall of the nasopharynx. These are the areas over which the exudate drips from the posterior sinuses, and the changes are apparently due to the enzymes in these exudates, or they may be due directly to offending allergens. In young children of allergic taint, particularly the tonsillectomized ones, the lymphoid hyperplasia becomes marked but is seen to involve the posterior wall of the pharynx rather than the lateral wall and accounts for the rapid regrowth of adenoids in many of them. One should be suspicious of allergy in every child presenting the so-called adenoid diathesis. Piness and Miller [4] have ably summed up their experiences in the following observations:

> . . . a regrowth of lymphoid tissue in the tonsillar fossae after tonsillectomy is as characteristic of allergy as the original enlarged tonsils, whose size alone so often seems to be the only indication which led to their removal. Lymphoid hyperplasia is one of the distinctive features of allergy.

FREQUENT COLDS

Of a series of over 1,200 patients whom we have attempted to immunize to the common cold because of their susceptibility to this disease, over 80 per cent have proved allergic or of an allergic family. Other observers have noted the incidence of colds in allergic persons.[5]

There have been attempts by others to differentiate the so-called allergic from the nonallergic cold.[5b, c] This has only resulted in confusion of nomenclature. Colds occur in allergic persons, but there is no scientific evidence that allergy causes the common cold. The numerous workers in the field of acute diseases of the upper part of the respiratory tract [6] have definitely described and identified the common cold and its

4. Piness, G., and Miller, H.: Allergy: A Nonsurgical Disease of the Nose and Throat, J. A. M. A. **85**:339 (Aug. 1) 1925; Allergy of the Upper Respiratory Tract in Infancy and Childhood. ibid. **113**:734 (Aug. 26) 1939.

5. (*a*) Hansel, F. K.: Further Observations on the Cytology of the Nasal Secretions in Allergy, read at the meeting of the Association for the Study of Allergy, San Francisco, Calif., June 12-13, 1938; abstracted, J. Allergy **10**:251, 1939. (*b*) Cohen, M. B., and Rudolph, J. A.: Allergic and Infectious Conditions of the Upper Respiratory Tract in Children: Differential Diagnosis, J. A. M. A. **97**:980-982 (Oct. 3) 1931. (*c*) Clein, N. M.: Allergy as the Cause of Frequent Colds and Chronic Coughs in Children, Northwest Med. **35**:347, 1936.

6. Foster, G. B., Jr.: The Etiology of Common Colds, J. A. M. A. **66**:1180 (April 15) 1916. Dochez, A. R.; Mills, K. C., and Kneeland, Y., Jr.: Studies on the Common Cold: Cultivation of Virus in Tissue Medium, J. Exper. Med. **63**:559, 1936. Shibley, G. S.; Mills, K. C., and Dochez, A. R.: Studies on the Etiology of the Common Cold, J. A. M. A. **95**:1553 (Nov. 22) 1930. Kruse, W.: Die Erreger von Husten und Schnupfen, München. med. Wchnschr. **61**:1547, 1914.

cause. The term allergic cold, appended to a train of symptoms characterized by sudden attacks of sneezing, watery discharge, nasal obstruction and eosinophilia, which is merely an acute manifestation of allergic rhinitis, is a misnomer and should be dropped from the vocabulary.

The most important accidental factor in infections is trauma. Immunologically speaking, in an allergic response, the union of antigen and antibody in the cells results in injury and release from the cell of some preformed histamine-like material. So, the stage is set in the nasal mucosa of the allergic patient through the continual bombardment by foreign proteins, causing injury by the release of histamine-like materials and the production of a locus minoris resistentiae. It may be said that physically normal persons who exhibit a tendency to frequent colds suffer from an allergic state, and, vice versa, the allergic patient suffers from frequent colds. It is normal for persons to get from one to three colds during a year. However, the sufferer from colds seeking help from the physician is likely to get from six to ten colds yearly, each cold occurring during one of the numerous cycles of virus colds which occur almost monthly throughout the year. Not infrequently, and particularly in children, these colds are so numerous that the patient continually has suppurative pansinusitis, which represents the residuum carried over from one cold to another. Such patients, it must be remembered, are physically normal, but if their noses are examined hyperplastic changes are found. Our belief that patients with frequent colds are allergic rests on the further evidence obtained by a survey of foreign proteins. Bacterial examination of material from the tissues of these patients reveals without exception, between attacks of colds, the organisms so frequently recognized as secondary invaders, namely, the pneumococcus, Friedländer's bacillus, M. catarrhalis, streptococci and B. influenzae, ready at the optimum time to overgrow. The optimum time apparently occurs after additional injury by the cold virus. These patients further show a decided cutaneous reaction to solutions of attenuated cold virus. Dochez and his colleagues [7] made the interesting observation that colds following the inoculation of virus in chimpanzees resulted in striking changes in the bacterial flora of the nose and throat. The pneumococci and influenza bacilli became much more numerous and appeared in the nasal passages which had previously been free of them as shown by culture. Most allergists [8] have noted that the hay fever sufferer who has numerous colds, after several years of treatment, even though he has not been desensitized to colds, exhibits only the number

7. Dochez, A. R.; Mills, K. G., and Kneeland, Y., Jr.: Filtrable Virus Infection of the Upper Respiratory Tract, J. A. M. A. **110**:177 (Jan. 15) 1938.

8. Sternberg, L.: The Effect of Pollen Injections on the Common Cold in Hay Fever Subjects, J. Allergy **6**:304, 1935.

of colds normally seen in nonallergic persons. It is recorded [9] that patients who have pollen hay fever also have symptoms during the pollen season from other allergens, to which they are not sensitive at other seasons of the year. This is one of the accepted reasons for failure to get results from pollen treatment when patients are not being desensitized to the offending allergens. The picture differs in no way in the patient sensitive to cold virus or its toxins. It must be appreciated, therefore, that of the numerous factors which predispose persons to colds, allergy is probably one of the most important, a factor long overlooked because of the failure to evaluate the condition properly during its formative stage. Unless the allergic state is eradicated, immunization to colds will continue to be the dismal failure it has been thus far, for the allergic person presents sensitization to the cold virus as well as to other allergens.

RECURRENT ACUTE LARYNGITIS, PACHYDERMIA LARYNGIS, SINGERS' NODES AND PAPILLOMAS

Laryngeal conditions which frequently are due to an allergic diathesis are recurrent acute laryngitis, pachydermia laryngis, singers' nodes and papillomas. These represent acute, chronic, diffuse and circumscribed hyperplastic changes in the tissues of persons who reveal similar changes throughout the nasal, sinal and pharyngeal mucosa. McKenzie,[10] in discussing the etiology of chronic laryngitis, stated that "the pachydermatous or hyperplastic type is often part and parcel of a constitutional tendency to fibrosis." It is not our contention, of course, that such conditions are always allergic, but it may be said that they are frequently seen in allergic patients.

Most investigators concede the frequency of pathologic changes in the nasopharynx in laryngeal conditions. Babbitt [11] makes the observation that acute laryngitis is usually seasonal, with regular recurrence in spring and autumn. This, of course, suggests a relation to the pollen seasons. We, too, noted this repetition of acute laryngeal conditions, but we also found them occurring in most allergic persons with each cold in the head, a concurrence which we noted particularly in borderline allergic patients not having manifest allergy or concomitant eosinophilia either in the secretions or in the tissues themselves. On the other hand, we have never been able to substantiate such conditions as autointoxica-

9. Surneford, O.: Physical Allergy, J. Allergy **6**:175, 1934.

10. McKenzie, D.: Diseases of the Throat, Nose and Ear, St. Louis, C. V. Mosby Company, 1928.

11. Babbitt, J. A.: Acute Laryngotracheitis, in Jackson, C., and Coates, G. M.: The Nose, Throat and Ear and Their Diseases, Philadelphia, W. B. Saunders Company, 1939.

tion, intestinal toxemia and defective elimination as etiologic factors in acute laryngitis, though these conditions are frequently mentioned as causing acute or chronic laryngitis.

Chronic laryngitis includes low grade inflammatory conditions which result in local or general hyperplastic changes throughout the larynx. Like hyperplastic disease of the nose, it is characterized by an overgrowth of all connective tissue, particularly in the areas most exposed to trauma by secretions, air or physical impact. These areas are, of course, the cords, the interarytenoid space, the arytenoids and the ventricular bands. Lederer [12] stated that the continuous postnasal dripping of irritating secretions into the pharynx and larynx predisposes these areas to hypertrophic and hyperplastic changes. This opinion is generally accepted by most laryngologists, but we feel that it is particularly true for persons having a hyperplastic diathesis, for they have like changes throughout the respiratory tract, from the uppermost nasal and sinal mucosa to the terminal bronchioles. This phenomenon may be compared to that noted in persons who are subject to keloids, except that the process occurs in a different system of the body. The absence of eosinophils in the affected tissues has perhaps caused observers to overlook the frequency of allergic taint in the patients. We must reconcile ourselves to the fact that only during an active allergic storm do eosinophils appear. Autopsy on material taken from hyperplastic areas throughout the larynx in persons who have died during an attack of asthma reveals an abundance of eosinophils.

The diagnosis of allergy must be made from a careful family and individual history, together with a complete allergic cutaneous survey. Typical cases demonstrating the conditions outlined are as follows:

Case 1.—L. D., aged 33, stated that as a child he had numerous attacks of croup following colds in the head. After reaching adult life he suffered constantly from a stuffy nose but never sneezed except with a cold. For the past ten years he has been subject to colds throughout the winter, lasting about two weeks and associated with hoarseness. Following his last cold he continued to be hoarse for five months. Except for a history of asthma in 1 parent, nothing else of significance was noted. Physical examination revealed hyperplastic changes throughout the nose, pharynx and larynx and a papillomatous growth about the size of a raisin on the left cord. This was removed and sectioned. The diagnosis was benign papilloma. A foreign protein survey revealed a 3 plus reaction to dust, ragweed, grasses, wool, extract of cold virus, flaxseed, beet, buckwheat, cheese and duck. The basal metabolic rate was —12 per cent; the chloride content of the blood was 432 mg. per hundred cubic centimeters, and the sugar tolerance was normal. Immunization to the materials mentioned, carried out weekly for one year, has resulted in freedom from nasal symptoms for the past eighteen months. Con-

12. Lederer, F.: Diseases of the Nose and Throat, Philadelphia, F. A. Davis Company, 1938.

current with this treatment the patient was given 1 grain (0.06 Gm.) of thyroid and 45 grains (2.9 Gm.) of sodium chloride daily. The patient was referred for instruction in the use of the voice and proper breathing.

CASE 2.—M. A., a girl aged 14, complained of hoarseness of six months' duration. A history of numerous colds since early childhood was given. She had had eczema in infancy. There was nothing else significant in the history except that the mother has suffered from headaches most of her life. Examination revealed hyperplastic changes throughout the nose and pharynx. Singers' nodes were present on the cords. A foreign protein survey revealed sensitivity (2 and 3 plus) to combined grasses, combined ragweed, dust, extract of cold virus, lemon, carrot, cucumber, walnut, veal, poppyseed, spinach, paprika, mushroom, celery, beef, tomato, olive, orange and green pepper. The basal metabolic rate was —20 per cent; chloride content of the blood was 442, and the sugar tolerance curve was normal. Progressive injections of the inhalant material were given weekly with 2 grains (0.13 Gm.) of thyroid daily. The child continued at school, using her voice. The voice became decidedly better after the eighth week and was normal after the twelfth week, although the former position of the nodes was just discernible. The treatment was continued for one year. There has been no recurrence to date, about two years since the beginning of treatment. Immediately after the disappearance of the nodes, the child was referred to a voice teacher for instruction in breathing and the proper use of the voice.

CASE 3.—Mrs. T. W., aged 57, had suffered from continuous loss of voice for three months. She had had asthma in childhood and suffered from about ten colds each year, always followed by attacks of laryngitis lasting approximately two weeks. Her mother was asthmatic. Laryngeal examination revealed uniform thickening, with numerous excrescences involving both vocal cords. Biopsy revealed chronic granulation tissue, i. e., hyperplasia. The diagnosis was pachydermia laryngis. Nasal examination revealed high grade hyperplastic changes throughout the nose and sinuses, with polypi in both middle meatuses. An allergic survey revealed sensitivity (3 and 4 plus) to ragweed, grasses, horse serum, wool, extract of cold virus, orris, camel dander, feathers, rabbit dander, cow dander, dust and pyrethrum. The patient was immunized to grasses, weeds, dust, wool and cold virus. After the third month the condition was greatly improved. The voice was normal after the fifth month. The patient has remained under treatment for a period of eighteen months, during which time her voice has remained normal.

CHRONIC PURULENT DISCHARGE FROM THE NOSE IN CHILDREN

Chronic purulent nasal discharge in children, associated with purulent pansinusitis, has long been the enigma of both pediatrician and rhinologist. Because of their habit of concentrating their efforts on the drainage of one or two particular sinuses, as is done for postscarlatinal conditions, rhinologists often forget to investigate the condition from its onset. However, in all the cases, if a careful history is taken one is impressed by the frequency of an allergic family history or even other allergic manifestations, particularly food problems in early infancy. The masking of the allergic condition by a chronic suppurative process is so complete as to make it impossible to diagnose it from physical findings or signs, so that dependence must be placed on a careful individual and family history. An illustrative case is as follows:

M. F., aged 5, according to his mother had presented a pediatric problem since early childhood, having had a purulent nasal discharge since he was 18 months old. He had always been a difficult feeder and had had bronchopneumonia twice and colds constantly throughout the year. He had had drainage of the antrum numerous times and his adenoids removed twice. There was a history of an aunt having hay fever and the maternal grandmother having asthma. Examination revealed highly thickened membranes throughout the nose, with much purulent exudate present. There were large amounts of lymphoid tissue in the pharynx. At the time of examination he was hoarse. A foreign protein survey revealed reactions (2 and 3 plus) to ragweed, grasses, dog hair, house dust, horse serum, lynx, mole, muskrat, rabbit, pyrethrum, lima bean, kidney bean, American cheese, Rocquefort cheese, corn, white potato, shrimp, tomato, veal, Monilia oidium, Monilia sitophilia and cold virus. Because of the patient's unruliness, it was impossible satisfactorily to carry out studies of the basal metabolism or of the sugar tolerance or the chloride content of the blood. The patient was immunized by weekly injections of ragweed, grasses, dust and cold virus and received weekly irrigations of the posterior sinuses. After five months of treatment the condition entirely cleared. The patient has continued to receive monthly treatments for the past year, without any recurrence of his former symptoms. It is significant that during the past epidemic of influenza all 5 members of his family were stricken, he and an aunt, who was also immunized, being the only ones to escape.

TUBAL-TYMPANIC CATARRH

Nonsuppurative conditions of the tubal-tympanic system are a natural sequence of pathologic changes in the nose and pharynx. The intimate connection of these systems through physical proximity, vascular and lymphatic continuity and similarity of protective mechanism causes them to share alike inflammatory processes which involve the membranes. The mucous membrane of the tube and middle ear is lined by a ciliated columnar epithelium, which, though slightly modified, is actually an extension from the nose and pharynx. It is a membrane rich in lymphatics and blood supply, constantly bathed by its own secretions and that from the nasopharynx. When the inflammation is acute the cells associated with a similar condition of the nose and pharynx are noted, for instance, round cells, histiocytes, fibroblasts and leukocytes. When the predisposing factor is an active allergen, eosinophils may appear throughout the mucosa. If, however, the activating allergens are mild and the condition goes on to chronicity, hyperplastic changes occur similar to those noted in the nose and pharynx. In the borderline allergic patient the changes in these membranes consist of thickening of the membrane due to increase in connective tissue elements and edema. The cilia are frequently lost, and metaplasia may take place. The predominant cell, as in the nasal and pharyngeal mucosa, is the round cell, with only a normal number of eosinophils. Only in an active allergic state do the eosinophils appear. It should not be difficult to understand, then, why about 25 per cent of patients with active hay fever suffer from symptoms referable to the tubal-tympanic system.

Proetz was quoted by Turner [13] as holding that in certain cases nonsuppurative otitis media is due to allergy. We have noted that about 65 per cent of our patients with hyperplasia, all of whom are borderline allergic patients, suffer from symptoms referable to the tubal or the tympanic region. This is particularly true of patients with long-standing hyperplasia who suffer from postnasal drip and intermittent obstruction, as described earlier in this paper. Stuffiness of the ears and tinnitus are the two most prominent symptoms of which these patients complain, symptoms which disappear when immunization is attained. Those in whom definite low tone deafness is proved by tests with the audiometer and the tuning forks, though immunization does not remedy this condition, react more rapidly and permanently to regular aural treatments such as catheterization.

TREATMENT

The treatment of borderline allergy is directed solely toward alleviation of the allergic diathesis. Surgical procedures and quasiscientific physical therapeutic methods, though seemingly of some temporary benefit, are in the end injurious, since they only result in fibrosis and a nervous and vascular chaos which ultimately causes a far greater vasomotor upset than was originally present. Along with the allergic study, a glandular survey should be conducted, including careful determination of the basal metabolic rate, the chloride content of the blood for insufficiency of the adrenals and the tolerance of sugar for pancreatic insufficiency and uterine scrapings for ovarian insufficiency. It must be borne in mind that, since the conditions are only mildly allergic, more concentrated materials must be used for testing. The danger of shock from the use of concentrated allergens is minimized in such patients because of the mildness of their condition and response to offending allergens. The use of concentrated allergens is likely to bring out a number of false positives, particularly among the foods. As in frank allergic conditions, however, they must be checked by trial in the diet to determine their clinical significance. It has been our observation that in adults the inhalants are preponderantly the greater etiologic factors in hyperplastic disease of the respiratory tract, while the converse is true in young children.

SUMMARY

1. The difficulties in properly evaluating the symptoms and pathologic changes of hyperplastic disease of the respiratory tract cause the borderline conditions to be falsely grouped in classifications which belie their allergic nature.

13. Turner, A. L.: Diseases of the Nose, Throat and Ear, Baltimore, William Wood & Company, 1936.

2. Neither the texture nor the color of the tissues nor the character of the exudate gives any hint of the allergic nature of the borderline condition, since pallor, edema and eosinophilia are usually not present.

3. Borderline allergy of the respiratory tract is diagnosed by the finding of gross hyperplastic changes in a person with a history of allergy or in whose family there is a history of allergy and in whom a foreign protein survey reveals positive sensitivity to particular allergens.

4. There is excellent evidence in many cases of the allergic nature of the following diseases when found in persons with hyperplasia:

A. Chronic intermittent nasal obstruction.
B. Chronic lymphoid pharyngitis.
C. Frequent colds.
D. Recurrent acute laryngitis, pachydermia laryngis, singers' nodes and papillomas.
E. Chronic purulent discharge from the nose in children.
F. Tubal-tympanic catarrh.

Limitation of space prevents publication at this time of any great amount of corroborative evidence, so that these observations must be considered a preliminary effort.

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SPEECH HEARING AND SPEECH INTERPRETATION TESTING

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The degree of deafness as determined by the audiometer, does not give even an approximate idea of the difficulty the patient may have in hearing the voice. It is the handicap for the voice that really counts. (Shambaugh Sr.)

After all, what we and the patient are interested in, is not the patient's ability to hear a tone reproduced through a telephone receiver, but his ability to hear the spoken voice. It is common knowledge that some patients' appreciation of the spoken voice is better than the audiometer record would lead us to believe, and that other patients' hearing for spoken voice is actually worse than the audiometer would lead us to believe. It is my opinion therefore that his audiometer tests leave the practical otologist dissatisfied. (Wishart.)

It must be remembered that the audiogram which is a guide to the threshold for sounds at various frequencies is no indication of the quality of perception at these frequencies. (Kerridge.[1])

Up to this time, audiometry has meant to most otologists the determination of pitch hearing. Yet, since there is little or no correspondence between the hearing for the various pitches and that for speech, there is a growing conviction that the only way one can determine speech hearing is by using speech as the testing stimulus. Any one working with the deafened is soon conscious that they are interested not in how well they hear pitches but in how well they hear the human voice. It behooves the otologist to pay more attention to speech hearing, since it is the concern not only of the patient but of the educator as well.

Certainly, the interest of laymen in the matter of hearing should be guided by otologists.

The only way the otologist can take his proper position is to obtain a thorough knowledge of the subject of speech hearing. To do this, he must become familiar with the acoustics of speech and the materials and methods available for testing the hearing of speech.

The acoustics of speech is outlined briefly in the following paragraphs:

OUTLINE FOR A STUDY OF THE HEARING OF SPEECH

1. The chief function of the organ of hearing is to hear speech.

Read at a meeting of the Philadelphia Laryngological Society, December 5, 1939.

1. Kerridge, P. M. T., in Ferguson, A.: Reports on Progress in Physics, London, Physical Society, 1939, vol. 5.

2. Speech and speech hearing are important.

3. A knowledge of the characteristics of speech and of the importance of the use and the phonetics of language should be the concern of otologists dealing with hearing.

4. The composition of language is as follows:

All language in any tongue is made up of vowel and consonant sounds. Vowels are more easily heard than are consonants, but proper consonant hearing is infinitely more important for the correct understanding of the word. In estimating the ability to interpret words, consonant hearing is given twice the value that vowel hearing is given.

5. All spoken language is characterized by the following factors:

Loudness of speech.
Speed of speech.
Voice register (high, medium or low pitched).
Sharpness of articulation or its opposite, elision.
Correctness in pronunciation.
Phrasing.
Accent.
Inflection.
Proper and improper use of words and grammar.

6. In relation to hearing, the spoken voice may be poorly heard for the following reasons:

(*a*) Speech may be too low.
(*b*) Speech may be too fast.
(*c*) Speech may be slurred.
(*d*) Pronunciation may be incorrect.
(*e*) The words used may be unfamiliar.
(*f*) Words may be used improperly in relation to meaning.
(*g*) Words may be used improperly as to grammatical construction.
(*h*) Pronunciation may not give the proper vowel and consonant sounds.
(*i*) Stuttering, stammering and lateral emission are obvious speech faults which make for difficulty in understanding. Dyslalia, the speech of the mentally retarded, or of patients with paralysis of the facial nerve, is difficult to interpret. Foreign accent is often as bad.

The English language is particularly difficult in three respects: 1. Words are not pronounced as they are spelled. 2. Words with different spelling may have the same pronunciation. 3. Words with the same spelling are pronounced differently.

EXAMPLES

Pair, pare, pear.
Mite, might; site sight.
Sley, slay; see, sea; *k*ind, *c*ake. } Pronounced alike but spelled differently
Dear, deer.
Dun, done.

(A foreigner following rules of pronunciation cannot understand why "done" is pronounced "dun" while "dome" is pronounced with a long "o.") *Close* to home, *close* the door—spelled alike but pronounced differently.

There is a greater list of vowels than one would suppose. A reference to a brief on the "Scientific Alphabet" (Funk and Wagnalls' Standard Dictionary, 1895, pages 2104-2107) will give an idea of the confusions and complexities which the language presents even for philologists.

EXAMPLE

There are varying pronunciations of the vowel "a"—far, fare, fat, partake, abase. This one vowel has been classified under types as follows: strong, variant, obscure, short, long.

The vowel "e" is "short, long, variant, obscure or silent."

The vowel "i" is short, long, obscure or diphthongal.

The vowel "o" is short, long, variant or obscure.

The vowel "u" is short, long, obscure or diphthongal.

If one considers that each of these variations means acoustically a distinctly different sound, one realizes that there are at least twenty-five different sounds representing simple vowels. The list would be greatly enlarged by adding all the diphthongs and the semivowel "y".

The consonant list is not so complicated, yet it too contains many more sounds as spoken language than the twenty-one consonant characters suggest. "K" is equal to "C" in most cases—cake, kind, cook, kite, cut, keen, cool, kill. This should reduce the count, but many consonants may be "hard" or "soft" or compounded into distinctly different sounds (ng, ch, st, tch, th). The combination gh may be silent, as in "through" and "though." How can one explain "enough" (enuff)?

Much conflict exists between spelling and pronunciation (ph = f [phonograph]). Sounds are often not spoken as spelled, nor spelled as spoken.

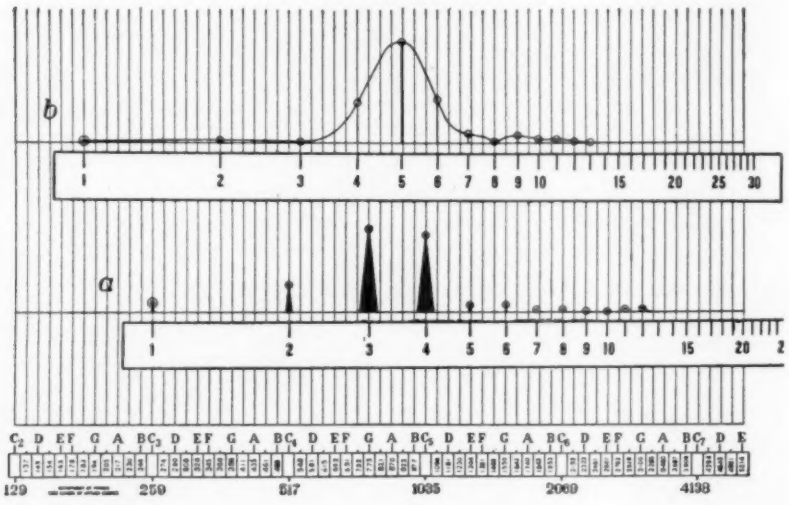
THE SIGNIFICANCE OF THE PHONETICS OF SPEECH TO THE OTOLOGIST

Speech represents an infinite variety of sounds or frequencies. Some are noisy; some are musical; some are explosive, sibilant, resonant, aspirated, nasal, soft, hard, loud, thin or rich in tone color. Each sound pattern has a meaning or significance. For each, there are a memory and an association.

One realizes the exquisite sensitivity of the hearing mechanism when it can receive, transform, amplify, transmit and perceive thousands of frequencies coming in the most rapid succession, as in speech.

Dayton C. Miller, in analyzing the vowel "a" as in "father," showed that sixteen major frequencies make up its character. One can then conceive that in a short spoken sentence there are thousands of pitches presented. Their interpretation and the dampening out of them must be practically immediate. It is understood, then, that such a finely functioning organ must be a most delicate, critical mechanism, as are most highly differentiated organisms.

It is surprising that the mechanism of hearing is not more often or more seriously damaged than it is.



The curve *b* represents an analysis of the vowel "*a*" in "father"; the curve *a*, an analysis of an organ tone, pitch 259. The numbers represent the number of overtones; for reference the harmonic scale is placed at the bottom of the diagram. (From Miller, D. C.: Science of Musical Sounds, New York, The Macmillan Co., 1922.)

A frequency analysis of the components of speech (illustration) is academically interesting, instructive and occasionally useful.

It seems, then, that with a knowledge of the frequencies of speech and with a record of a patient's hearing across the frequency range one might deduct much. Harvey Fletcher proposed the averaging of the middle frequencies as an index of speech hearing, for the important characteristics of speech lie in that zone. (Parenthetically, it is interesting to note that the "macula" of hearing lies in the same zone.)

On the Western Electric audiogram chart there is a footnote that the percentage of loss of hearing for speech is approximately equal to the average reading at 512, 1024 and 2048 multiplied by 0.8. This statement has unfortunately been accepted by many unfamiliar with testing for speech hearing. A large series of audiograms and of tests of speech hearing on patients shows that one cannot infer from an audiogram the efficiency of speech hearing.

The comparative findings from a summary of the records of 22 patients tested by the 2-A (pitch range audiometer) and the 4-A (phonograph) instruments are listed as follows. The records have been translated into speech hearing by the Fletcher method (the average of the loss in decibels for the frequencies 512, 1024 and 2048 multiplied by 0.8 equals the percentage of speech hearing). The numbers indicate percentage of loss of hearing.

2-A Record (Pitch Range Audiometer)	4-A Record (Phonograph)
4	00
24	12
18	00
20	9
22	6
8	6
48	30
16	9
25	12
10	0
9	6
46	3
13	15
24	15
26	15
22	12
44	18
9	3
34	18
10	0
12	0
45	12
489	201

The 22 comparisons between the two methods show that there is a variation in the order of 22:9, the 2-A method showing higher losses of hearing.

METHODS AND MATERIAL

Speech hearing can be tested only by the use of speech as the test stimulus.

The motor-driven phonograph, with its electrical pick-up and decibel meter, has revolutionized testing for speech hearing. With this instrument, any desired intensity may be obtained and repeated with identity. The word lists and the material used in the records should be selected with great care, as to articulation, quality of recording, needle scratch and familiarity of the words used. I have found the following word list useful. It represents a selection of 50 monosyllables from the words

most commonly used in the English language, based on Gate's list of the first 50 words in frequency of use.

The	Make	We	Get	Had
I	One (adj.)	It	Two	Your
Is	One (noun)	Me	His	Give
To	Do	Of	Are	Mother
In	For	My	Her	Up (prep.)
You	Be	Boy	Play	Up (adv.)
He	All	Our	Day	Home
On	See	Man	Have	Made
No (adv.)	Go	Come	Big	Red
No (adj.)	At	A	Run	Eat

The frequency of the various vowel sounds in the list is as follows:

ĕ.....5	oo.....4	a (make).....5	ou.....1
ē.....7	o (on).....6	a (all).....2	u.....3
i.....2	o (no).....4	a (at).....4	u.....2
u 6			

As familiarity with words largely determines their interpretability, a still larger list is important to consider and to use for testing words which appear more frequently in speech. Much work has been done in actually counting the instance of various words, and graded vocabulary lists have been made showing the size of the vocabulary for children of different ages. Naturally, the vocabulary of the adult depends on his education. In the following abstract from Thorndyke's classic list are monosyllables selected from the first 500 words in frequency of use in the English language, from a list of 4,265,000 words. These monosyllables comprise 90 per cent of the list. These words are familiar to all persons over the age of 6. The list is useful in investigating the size of the hearing vocabulary of the deafened child and may also be used in teaching this child the important words in the language.

(A)	Come	Food	How	Men	Rain	South	Town
Add	Corn	Foot	(I)	Might	Raise	Speak	Train
Air	Cross	(For)	If	Mill	Reach	Spring	Tree
(All)	Cut	Form	(In)	Milk	Read	Stand	True
Am	Dark	Four	Is	Mild	Red	Start	Try
An	Day	Free	It	Mine	Rest	State	Turn
(And)	Dead	Fresh	Its	Miss	Rich	Stay	To
Any	Dear	Friend	Just	Mouth	Ride	Step	Up
Are	Death	From	Keep	More	Right	Still	Us
Arm	Deep	Front	Kill	Most	Road	Stone	Voice
(As)	Did	Full	Kind	Move	Rock	Stop	Wait
Ask	Die	Gave	King	Much	Roll	Story	Want
(At)	Do	Get	Know	Must	Room	Street	Walk
Back	Does	Girl	Known	My	Round	Strong	Wall
Bad	Done	Give	Land	Name	Run	Such	Wax
Ball	Door	Glad	Large	Near	Said	Sun	Warm
Bank	Down	Go	Last	Need	Sail	Sure	Was
(Be)	Draw	God	Late	New	Same	Sweet	Watch
Been	Drink	Gold	Laugh	Next	Save	Take	Way
Bed	Drive	Great	Law	Night	Saw	Talk	We
Best	Drop	Green	Lay	No	Say	Tell	Week
Big	Each	Grow	Lead	North	Sea	Ten	Well
Bird	Ear	Had	Learn	(Not)	See	Than	Went
Bear	Earth	Hair	Leave	Now	Seem	Thank	Were
Black	East	Half	Left	(Of)	Send	(That)	What
Blow	Eat	Hand	Length	Off	Sent	(The)	When
Blue	Egg	Hard	Less	Old	Serve	Their	Where
Body	End	Have	Let	(On)	Set	Them	Which
Book	Eye	Has	Lie	Once	Shall	Then	While
Both	Face	He	Life	One	She	There	White
Box	Fair	Head	Light	Or	Ship	These	Who
Boy	Fall	Hear	Line	Our	Short	They	Whole

Bring	Far	Heart	Live	Out	Should	Thing	Why
Build	Fast	Help	Long	Own	Show	Think	Wide
Burn	Fear	Her	Look	Part	Side	Third	Wind
But	Feel	Here	Lost	Pass	Sight	This	Will
By	Feet	High	Love	Pay	Since	Those	Wish
Buy	Few	Hill	Low	Piece	Sing	Though	With
Call	Field	Him	Made	**Place**	Sit	Thought	Wood
Came	Fill	His	Make	Plain	Six	Three	Word
Can	Find	Hold	**Man**	Plant	So	Through	**Work**
Care	Fine	**Home**	Many	**Play**	Soft	Till	**Year**
Cause	Fire	Hope	Mark	Please	Some	**Time**	Yet
Child	First	Horse	May	Point	Son	(To)	You
Clear	Five	Not	Me	Poor	Soon	Too	Young
Close	Floor	Hour	Mean	Put	Sound	Top	Your
Cold	Fly	House	Meet	Quick			

The 6 words in bold face type are the most commonly used nouns. The words in parentheses are the 15 most commonly used words. Other useful records are based on lists of consonants and vowels.

Tool	Boot, boon	What is a tool?
Took	Book, buck	He took the train
Tone	Bone, boat	Do you like the tone?
Talk	Bought	Can animals talk?
Ton	Bog, box	Coal is sold by the ton
Top	Bar, bark	The boy plays with his top
Tap	Back, bat	I heard a tap
Ten	Bet, beck	He gave her ten
Tape	Bake, bait	He wrapped it with tape
Tip	Bit, bin	It was on the tip
Team	Beat, bean	John made the team
Time	Bite, bike	What is the time?

CONSONANT LIST FROM THE PHONETIC ALPHABET (110 WORDS)

Bed	Bird	Black	Book	Box
Day	Dark	Deep	Did	Die
Feet	Fire	Floor	Far	Feel
Girl	Go	Green	God	Get
Keep	Kill	King	Kind	Can (K)
Late	Long	Live	Look	Light
Man	Me	Milk	Mouth	More
Not	Now	No	Night	New
Red	Read	Rain	Ride	Road
Place	Pay	Plant	Poor	Put
Said	Saw	See	Send	Sit
She	Ship	Short	Show	Shall
This	Those	That	The	Them
Three	Through	Think	Thing	Thank
Time	To	Top	Tree	Try
Voice	(Vie)	(Vote)	(Vine)	(Vim)
Church	Child	(Choose)	(Shum)	(Chop)
(Zee)	(Zoom)	(Zip)	(Zee)	(Zoom)
Just	(Judge)	(Joke)	(Jack)	(Jake)
Hand	Hard	Head	Help	Her
Warm	Wait	Want	We	Walk
Year	Yet	You	Young	Your

These words, except those in parentheses, are monosyllables in the list of the first 500 in common use.

Help in Choosing a Hearing Aid.—One approach which the otologist can make toward breaking down the barrier between himself and the deafened is the service he can give in the selection of a hearing aid. The aids are relatively expensive for many such persons, and they are often suspicious, both justly and unjustly, of the salesman's desire to make a sale. The otologist may act as an impartial advisor as to whether a new aid will help hearing or whether it is better than the old aid.

With a set of speech records and a phonograph with a loud speaker, one may try out the hearing of the patient with and without his aid. With such equipment, one may determine what vowel or consonant sounds are most difficult for the patient to hear; one can tell how much intensity of sound is needed and whether low or high tones of speech should be dampened out.

Determining if Hearing is Improved by Suppressing Low Tones.—Every one is familiar with the change in pitch which occurs when a phonograph record is speeded up; music and speech both become more thin and squeaky.

Test: The threshold intensity for speech hearing is determined with the record running at the normal speed, 78 revolutions per minute. The intensity is turned down to a point at which the patient can hear 1 out of 5 of the test words correctly. Then the record is gradually speeded up. The low tones in the speech become less prominent; the pitch of the voice rises. Though the test words come more rapidly and give less time for the patient to interpret, most of the deafened show an increase in the number of correct responses. One knows, then, that in this case a hearing aid picking up or exaggerating low tones is neither needed nor desirable.

Determining if Hearing is Improved by Suppressing High Tones and by Increasing Low Tones.—By reversing the foregoing method, the patient is first tested with a record turning faster than normal. The intensity is reduced until an occasional word is answered correctly (1 out of 5). The speed of the record is then reduced to normal, and the patient's hearing either improves or is worse, as shown by the number of correct answers. (A stroboscopic disk placed on the turntable determines the normal speed of the record, 78 revolutions per minute. These disks may be obtained from any large phonograph agency.)

Testing the Speech Hearing of Very Young Children.—With young children, Crowden's method is used. A series of words which represent the simplest and first-learned words in the language is selected and recorded. With a picture card in front of him the child can point out the object named. The test becomes a game to the little patient, and he is eager to make a correct performance. The method is much more accurate with timid children than if they had to speak out their answers.

Judging Central Perception.—Apart from the ability shown by a patient in hearing a phonograph record of simple speech sounds, there is with every person a certain degree of agility in central perception. Two persons may show identical hearing with the phonograph audiometer, both requiring the same decibel amplification for correct

reporting, yet one of them may have much more difficulty in hearing conversation than the other. Why?

The answer is not easy, for a number of factors take part, as in the following illustration: Two persons have the same amount of distortion of hearing; although their frequency audiograms show their distortions to be at different frequency levels, they report on the simple speech record at the same amplification. But when listening to conversation, one person shows confusion and difficulty; the other does not. The mental attitude plays an important part.

Many a deafened person fears to appear confused, dull or stupid. The fear itself brings more confusion and possesses the person to such an extent that he is distracted from listening intently. At times he has picked up a few words which have led him to believe that the subject matter is following a certain line. Then, suddenly, he hears a word that does not possibly fit into the trend. For example, "My *brother* is very fond of a *pipe*, but he throws matches *all over the house*." Only the italicized words were heard, and some meaning has been obtained from the first part of the sentence by the hearing of the two words "brother" and "pipe." But what can the phrase "all over the house" mean, when it is standing alone? "It must have something to do with "pipe" or "brother," but what"? If he stops to wonder, the next sentence in the conversation is on him, and he is not ready for it. (It is obvious from this why one gives the deafened person a better chance if one speaks slowly as well as distinctly).

Familiarity with phrases and forms of speech undoubtedly plays a part in central perception. Practice in listening exalts this ability.

General knowledge and a good education should improve the agility in correctly interpreting what is partially heard.

Attention is a most important factor in central perception. There are all degrees of it with the deafened. Some show a quick arousing and a sharp focusing; others have to have their hearing warmed up by speech louder than necessary. Some, it seems, have to be shaken out of their lethargy or their absorption.

Testing Hearing Agility.—I think it is well to test the patient first for the sharpness of the hearing threshold, which is itself a good index of agility. This testing is done by recording the threshold when going from inaudible to audible and then recording the threshold in going from audible to inaudible. If the two thresholds match each other or are near each other, good acuity may be anticipated. (The "audible to inaudible" should give the lowest threshold, yet care should be taken when one is working with tones instead of words that the patient does not develop tone memory and thus mentally carry the tone down. One

should use the sound stimuli irregularly on and off or swing the pitch back and forth in the zone of the frequency tested.)

Seashore's records for testing musical talent are certainly tests in the perceptive or interpretive spheres rather than tests of gross hearing ability. This is particularly true of the test for the discrimination of intervals (time) between notes. Pitch discrimination tests, on the other hand, do not seem to correlate with agility, nor does the test for dissonance or consonance. The tone memory test tends to show the best results in those with good auditory agility.

I have devised a test which promises fairly dependable quantitative data and is simple and rapid. A series of 10 carefully chosen words is recorded in successive bands on a record. The sequence is scrambled for each band, but the words are the same. The interval between the individual words on the bands is 10, 8, 6, 4 and 2 seconds for the respective bands. The intensity level of the record is set for convenient hearing, and this loudness is recorded in the reporting along with the loss of speech hearing decibels. Age, of course, is a noticeable factor in this test, as Seashore found in his tests, and a child's agility must be judged with consideration of the "rough-normal" for his age. It is obvious that those with poorest agility will not be able to report on the short interval bands as correctly as on the long interval bands.

QUESTIONS AND ANSWERS

Q. What is meant by the term articulation test as applied to hearing?

A. Articulation testing is the determination of the ability to recognize speech sounds. If mistakes are made in interpreting correctly the spoken word or syllable, it may be "desirable to further analyze these mistakes and determine the articulation of speech sounds. The percentage of the total number of spoken sounds which are correctly observed is called the *sound articulation.* When the attention is directed toward a specific fundamental sound, such as 'b' or 't' or 'a,' etc., then the term *individual sound articulation* is used. For example, the individual sound articulation for 'b' is the percentage of the number of times that 'b' was called that it was observed correctly. Similarly, the terms *consonant articulation* or *vowel articulation* refer to the percentages of the total number of spoken consonant or vowel sounds which are correctly observed." [2]

Q. When English words and short sentences are used as testing stimuli, what should this testing be called?

2. Fletcher, H., and Steinberg, J. C.: Articulation Testing Methods, Bell Telephone Laboratory. This article is the most comprehensive one on the values of speech sounds in the literature and will be of inestimable value to any person making a study of the subject.

A. Fletcher prefers and uses instead of the terms "word articulation" and "sentence articulation," the terms "discrete word intelligibility" and "discrete sentence intelligibility," since a new element enters, namely, the thought or meaning associated with the sentence or word.

Q. How much better should numbers be heard than common words?

A. Numbers are usually heard or recognized 10 decibels lower than words.

Q. To hear sentences perfectly, what must be the percentage of correct word hearing?

A. Articulation has to be 70 per cent efficient to obtain perfect sentence intelligibility, yet a mentally agile deafened person can understand correctly most of what is said with considerably lower performance in articulation.

Q. Does the patient's hearing have to be "warmed up" by listening to readily audible sounds before the best threshold showing can be made?

A. In many cases it does; this is especially true when some degree of attention deafness has become established.

Q. Are meaningless speech sounds practical for everyday testing by the otologist?

A. I believe that they are most impractical, particularly from the points of view of reporting (repeating), recording and grading.

Q. Are sentences useful in testing?

A. Not for quantitative measurement of speech hearing; intelligence, education and familiarity with phrases and ideas play too large a part. For example, "He took the train"; "What is the time?"; "Coal is sold by the ton." The correct words may too often be guessed.

Q. Does the needle scratch interfere with phonographic testing of speech hearing?

A. No, not appreciably. The modern light weight and tangential tone arm has reduced the scratch to a negligible factor.

Q. By auricular (central acuity) training may intelligibility be indefinitely improved?

A. Not indefinitely nor infinitely improved, yet even a 10 decibel improvement may make the difference between understanding no speech and understanding a considerable amount.

Q. Can the phonograph be used as a training instrument for the little child, deaf before acquiring the use of language, who has some hearing?

A. The phonograph is a distinctly helpful adjunct to the established methods of reaching and teaching language to the deaf child.

CONCLUSIONS

The testing of speech hearing must be approached with an understanding of the characteristics of speech.

The patient is primarily interested in speech hearing.

There is no correlation between speech hearing and frequency hearing.

Speech hearing can be tested only by the use of speech as a testing stimulus.

The phonograph should be used in place of the old test with the spoken voice.

A knowledge of testing for speech hearing and the proper equipment should be in the armamentarium of every otologist.

1805 Chestnut Street.

Case Reports

NASOPHARYNGEAL CARCINOMA

Extension Intracranially and Metastasis by Implantation into the Soft Palate; a Clinical and Pathologic Study

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The following case of carcinoma of the nasopharynx presents several features interesting not only to the otolaryngologist but also to the neurologist, the ophthalmologist, the roentgenologist and the pathologist. It is presented because of its value to both the clinician and the pathologist in that an attempt has been made to explain clinical symptoms from a complete study at autopsy.

Of further interest was the intracranial extension of the tumor with marked destruction of bone, metastasis to the liver and an apparent implantation of tumor cells by contact from the growth in the nasopharynx to an ulcer on the nasal surface of the soft palate. This ulcer approximated and was irritated by the tumor in the nasopharynx on phonation and swallowing.

That malignant tumors of the nasopharynx are not uncommon is evidenced by the abundance of literature on the subject, many authors reporting fairly large series of cases. It was our good fortune to study more minutely the pathologic anatomy of a case which we had observed clinically and in which we were allowed unrestricted material for autopsy.

Trotter [1] stressed the nasopharynx "a region rich in pathologic interest and subject to the growth of numerous tumours, which are by no means infrequent and are of great diagnostic importance." He directed attention to that class of growth which produces little or no projection into the nasopharynx and is therefore dismissed as having little diagnostic interest and to the tumor which projects into the nasopharynx and gives rise to obvious symptoms. He particularly stressed the importance of recognizing the former and stated that "diagnosis is entirely dependent upon recognition of the symptoms of infiltration of the naso-pharyngeal wall. . . . The strictly anatomical distribution of the symptoms is always a striking feature." He emphasized a triad of symptoms which should focus attention to such lesions of the nasopharynx, viz.:

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From the Department of Pathology, Cook County Hospital, and the Department of Laryngology, Rhinology and Otology, Research and Educational Hospitals, University of Illinois College of Medicine.

1. Trotter, W.: On Certain Clinically Obscure Malignant Tumors of the Nasopharyngeal Wall, Brit. M. J. **2**:1057 (Oct. 28) 1911.

1. Pressure on the eustachian tube, causing impairment of hearing.
2. Pain of the trigeminal nerve, especially of the third branch.
3. Asymmetry of the soft palate due to the pressure of a large mass or to direct involvement by the tumor of the levator palati muscle.

The groupings of symptoms which occur may be divided according to whether they indicate involvement of the respiratory tract, the ear, the nerves, the eye or the body as a whole or whether they arise from metastases. Closure of the jaw is usually a late manifestation. Enlargement of the cervical glands is a constant feature in late phases.

New [2] called attention to remote symptoms referable to one ear, diplopia or regional metastases as common early signs. Woltman,[3] in reporting 79 cases, stated that the cranial nerves are usually involved extracranially. Those passing through the orbital fissures are most commonly affected, particularly the sixth, and, next most frequently, the trigeminal.

Hansel [4] in reporting 17 cases, remarked that comparatively few cases have been reported in which the diagnosis was based primarily on involvement of the nervous structures. Needles [5] discussed the tumors with special reference to the neurologic complications, emphasizing the failure to establish an early diagnosis, and stated, "Unless, therefore, when confronted by a basilar syndrome, one associates it with a possible focus in the nasopharynx, the diagnosis may be overlooked." In 16 of his 35 cases neurologic involvement was evidenced. The most common symptoms were sensory disturbances of one side of the face, oculomotor disturbances, ptosis and diplopia. The cranial nerves usually affected were the fifth, sixth, third and fourth. If the tumor invades the skull, it tends to remain extradural. Furstenberg,[6] in describing 40 cases of malignant neoplasms of the nasopharynx, stated:

> It is a noteworthy fact that these neoplasms show a striking tendency to infiltrate upward, erode bone, and eventually disturb those structures which pass through the basal foramina. . . . Of practical importance, therefore, is the observation that these new-growths often assume enormous proportions and consequent destructive behavior long before there is unmistakable evidence of their existence.

The most common symptoms which he noted were pain in the head, ear or throat, cervical swelling, deafness or fulness of the ear, nasal obstruction, nosebleeds, double vision and paralysis and swelling of the face. Hauser and Brownell [7] presented a series of cases, detailing a group of symptoms and signs which often lead to correct diagnosis,

2. New, G. B.: Syndrome of Malignant Tumors of the Nasopharynx, J. A. M. A. **79**:10 (July 1) 1922.

3. Woltman, H. W.: Malignant Tumors of the Nasopharynx with Involvement of the Nervous System, Arch. Neurol. & Psychiat. **8**:412 (Oct.) 1922.

4. Hansel, F. K.: Malignant Tumors of the Nasopharynx with Involvement of the Nervous System, Ann. Otol., Rhin. & Laryng. **41**:74 (March) 1932.

5. Needles, W.: Malignant Tumors of the Nasopharynx, J. Nerv. & Ment. Dis. **86**:373 (Oct.) 1937.

6. Furstenberg, A. C.: Malignant Neoplasms of the Nasopharynx, Surg., Gynec. & Obst. **66**:400 (Feb.) 1938.

7. Hauser, J., and Brownell, O.: Malignant Neoplasms of the Nasopharynx, J. A. M. A. **111**:2467 (Dec. 31) 1938.

and emphasized that the nasopharynx should be given careful and routine inspection by the rhinologist.

Schlivek [8] from his study of 38 cases concluded that involvement of the fifth, sympathetic and sixth nerves, either individually or in combinations, with symptoms referable to the ear, is strongly suggestive of nasopharyngeal tumor and that the presence of signs of involvement of the eye indicates that the tumor has already invaded the skull.

These, as well as other authors, including Martin,[9] have pointed the way to early recognition of malignant growths of the nasopharynx. We believe that such a growth should be strongly suspected in the presence of neurologic symptoms, such as headache, trigeminal neuralgia, paralysis of the abducens nerve, diplopia, oculomotor or trochlear paralysis and defective hearing. These signs, together with an associated conductive type of deafness, can lead to the correct diagnosis. Unfortunately, even at such a stage, the prognosis is usually poor and treatment unsatisfactory. Swelling of the cervical glands may occur early but usually is a late manifestation.

REPORT OF A CASE

History.—A. P., a white man aged 49, employed as an engineer, first presented himself at the Research and Educational Hospital in July 1938 with the following complaints:

1. Headaches and dizziness of three years' duration.
2. Epistaxis of two years' duration.
3. Diplopia of nine months' duration.
4. Internal strabismus of the left eye of nine months' duration.
5. Internal strabismus of the right eye of four months' duration.

The past history was noncontributory except for a nasal and postnasal discharge of several years' duration.

The family history was essentially irrelevant except for one brother having died at the age of 36 of a sarcoma.

Present Illness.—Three years previously the patient had consulted his family physician, complaining of epigastric distress, nausea and vomiting, dizziness and dull frontal, temporal and occipital headaches. The nausea and vomiting occurred about one to two hours after meals. Alkaline powders were prescribed, and the gastric symptoms were relieved, but the headaches and dizziness persisted, becoming more frequent and severe.

Two and one half years previously he had noticed ringing in both ears; at the onset the ringing was intermittent, but during the last nine months it had been continuous. Two years previously he began to have nosebleeds, which continued intermittently and were often severe until death.

Nine months previously, while shaving, he noticed that he had difficulty in placing the razor against his cheek because of double vision and turning in of his left eye.

He consulted an "eye doctor," who after treating him for three months referred him to the Illinois Eye and Ear Infirmary (six months previously). Roent-

8. Schlivek, K.: Ocular Manifestations of Malignant Nesopharyngeal Tumors: Report of Cases, Arch. Ophth. **17**:1055 (June) 1937.

9. Martin, C. L.: Complications Produced by Malignant Tumors of the Nasopharynx, Am. J. Roentgenol. **41**:377 (March) 1939.

genograms were taken of his head, and he was informed that he had "sinus trouble." Treatment consisted of irrigation of his sinuses and roentgen therapy, presumably to his sinuses. He was also given glasses, which helped his diplopia and enabled him to read for short periods. When he was refitted for glasses three months previously, it was noted that he had internal strabismus of the right eye also.

The patient had also complained of dyspnea for the past two months, as well as nocturia which disturbed him three to four times a night, and generalized muscular weakness for the past six months. He had lost 30 pounds (13.6 Kg.) since the onset of his present illness.

He was not improving; so he was admitted to the neurologic service of the Research and Educational Hospital on July 6, 1938, three years after the onset of his symptoms. He arrived with a slip stating that examination of the visual fields, eyegrounds, spinal fluid and blood gave negative results.

Physical Examination.—The pulse rate was 80; the blood pressure, 120 systolic and 70 diastolic. When admitted to the hospital in July 1938, the patient was a well developed, thin white man, about 50 years of age, apparently not acutely ill. His temperature and pulse rate while he was in the hospital were normal.

His skin was smooth and warm; seborrheic dermatitis was present over the sternum and back. The ears did not evidence tenderness over the mastoid, and the drums were retracted. The mouth showed numerous carious teeth. A postnasal discharge was present on the posterior wall. The neck was normal, and there were no palpable glands. Involvement of the first cranial nerve was evidenced by bilateral anosmia. That of the second was indicated subjectively by failing vision, with roughly no limitation of the fields, while objectively the fundi were normal. Examination of the third showed normal convergence and vertical movements; the pupils were equal and round and reacted sluggishly to light and in accommodation, and vertical nystagmus was seen in the right eye. The fourth and fifth were normal. Involvement of the sixth was suggested by bilateral internal strabismus. The seventh was normal. Examination of the eighth showed the subjective symptoms of tinnitus of two and one-half years', and diminished hearing of one year's, duration, while objectively it was noted that the patient heard a watch tick only when it was placed against his ears and that the bone conduction was better than the air conduction bilaterally. The ninth, tenth, eleventh and twelfth nerves were normal. The superficial and deep reflexes were normal. The chest was emphysematous, with increased tympany and fremitus and dry rales at the bases of both lungs. The heart, abdomen, trunk, extremities and rectum were essentially normal. Abnormalities of gait, posture or habitus were not seen.

In summary, the patient complained of headaches and dizziness for the past three years and diplopia for nine months. The only positive findings were bilateral internal strabismus, middle ear deafness and chronic pansinusitis.

The impression was that of a lesion of the sixth cranial nerve, which might be attributed to localized meningitis or to syphilis.

Laboratory Examination.—The urine was essentially normal. The erythrocyte count was 4,050,000, with hemoglobin 11 Gm. per hundred cubic centimeters; the leukocyte count, 10.570, with lymphocytes 12 per cent, monocytes 1 per cent, neutrophils 85 per cent and eosinophils 2 per cent. The spinal fluid was under a pressure of 140 mm. of water and clear, with a cell count of 3 (all lymphocytes), a negative Wassermann reaction and a 0000000 reaction to the colloidal gold test. Chemical analysis of the blood showed dextrose 83 mg. per hundred cubic centimeters, nonprotein nitrogen 35.6 per hundred cubic centimeters and a carbon dioxide–combining power of 64. The Wassermann and Kahn reactions of the blood

were negative. On roentgen examination the petrous tip showed, in Stenvers' position, no pathologic change. The skull viewed stereoscopically in the right lateral position showed no definite pathologic change. The sinuses revealed bilateral pansinusitis.

Course.—The report of a neurologic consultation July 8 stated: "The patient presents almost complete bilateral palsy of the sixth nerve. In looking upward with the right eye covered he experiences horizontal nystagmus of the left eye. There is no sense of smell, perhaps because of surgical intervention. The visual fields are roughly normal; the pupils are normal, and there is apparently no involvement of the third cranial nerve. All other cranial nerves are normal, except that there is obstructive deafness on both sides. The central nervous system is otherwise normal, except for rather brisk tendon reflexes. No pathologic reflexes are present. The diagnosis is a malignant growth with metastasis or some progressive lesion of the brain stem."

An otorhinolaryngologic consultation July 14 was reported as follows: "The nose shows thickening of the septum and deviation to the left. There is pus in both middle meatuses. The teeth are carious. The pharynx is small, with embedded tonsils. The ear drums are dull, gray, thickened and retracted. The hearing time measured by the Schwabach test is slightly diminished. The reaction to the Rinne test is bilaterally negative. Perception of low tones is poor; that of high tones, slightly diminished. A paretic type of ocular nystagmus is present, but no vestibular type. Spontaneous past pointing does not occur. A good caloric response is seen on both sides in nystagmus and past pointing, but function is not induced in the paralyzed external rectus muscles. No vertigo or vasomotor reaction is induced by vestibular testing. The diagnosis is a probable nonsurgical basal lesion."

The patient was discharged on July 16 with a tentative diagnosis of bilateral paralysis of the abducens nerve of unknown cause. He was advised to return at frequent intervals to the dispensary for further examination.

On August 15 no change was recorded.

On August 31 a flare-up of "sinus trouble" and also an increase in the severity of the neurologic symptoms were noted. The right eyelid was then drooping so far that the eye was closed. The patient had pain in the left side of the neck and face.

On September 8 there had been definite progress in the patient's condition; he then had complete oculomotor paralysis on the right, with drooping of the lid and inability to move the right eye in any direction. The patient was readmitted to the neurologic service of the hospital, where he was kept under observation until October 6, at which time he was again discharged with the following summary:

"This 49 year old white man was last in the hospital on July 16, 1938. At that time his condition was diagnosed as bilateral paralysis of the abducens nerve, possibly on an encephalitic basis. He has returned now because of complete paralysis of the third nerve on the right side. He has had constant severe headaches in the frontal region, and, in addition, he has a marked nasal and postnasal discharge. Roentgenograms revealed marked sinusitis, and he was given high voltage roentgen therapy with some relief and with some improvement of the paralysis of the third nerve. He is discharged with instructions to return to the dispensary for further observation and high voltage roentgen therapy."

On November 11 he was examined in the otorhinolaryngologic dispensary, and the following report was made:

"There is complete oculomotor paralysis on the right, with no reaction of the pupil, and also paralysis of the abducens nerve bilaterally. There is secretion on

both sides of the nose. The tongue can move to the left from the midline but not to the right. A large, firm gland is palpable on the left side of the neck.

"Massive irrigation of the left ear produces dizziness; the left eye shows typical nystagmus to the right; the right eye, only slight nystagmus to the right. Massive irrigation of the right ear produces dizziness and double vision, which was not present before. The left eye moves to the right and shows a rough rotary nystagmus to the left; the right eye does not move but reveals slight nystagmus to the left. Turning to the right produces dizziness, typical falling and nystagmus similar to that produced by irrigating the right ear.

"Examination of the nasopharynx reveals granulation masses surrounding the left eustachian orifice, almost hiding it from view. A specimen for biopsy was taken from this area.

"Examination of the larynx shows chronic laryngitis. The larynx and the pharynx are totally anesthetic. The patient has some degree of ankylostoma."

On November 29 a biopsy of the tumor in the nasopharynx was reported on as follows: "There are several fragments which include: (*a*) part of a nasal polyp revealing a folding-up of mucosa with hyperplastic transitional changes from tall columnar to pseudostratified epithelium, which has extensive inflammatory reaction and fibrosis (nasal part); (*b*) an adjacent area covered by thickened, semikeratotic squamous epithelium, which is everywhere demarcated. At one end of this fragment are massive strands of small, irregular flat cells. Whether these originated at the pharynx or from the nasal portion cannot be definitely decided from these fragments, but they are nevertheless malignant. The diagnosis is transitional cell carcinoma."

The patient was given high voltage roentgen therapy directed toward the nasopharynx and the glands on the left side of neck. He continued to have pain on the left side of his face.

On December 30 the patient had a severe spontaneous hemorrhage from the nasopharynx and was admitted to the Cook County Hospital, where he died the same day.

Autopsy (Dec. 31, 1938).—There was a grayish white tumor the size of a walnut in the right lobe of the liver and a smaller one in the left lobe. On gross sectioning these appeared grayish white, the larger containing a small cyst filled with blood-stained fluid. The brain appeared to be slightly compressed over the anterior inferior aspect; the meninges were thin, the blood vessels thin walled and patent and the convolutions flattened. The walls of the infundibulum were thinned, and there was slight internal hydrocephalus, especially on the left side.

On removal of the brain from the skull, the sella turcica was seen to be enveloped by a grayish white tumor, which was moderately firm, measuring 60 by 42 by 30 mm. By a method which allowed removal without collapse of the head, the base of the skull, including the nasopharynx, was removed, as were the soft palate, the tongue and the pharynx. In the nasopharynx, just beneath the sella turcica, a grayish white, moderately firm tumor was observed.

The removed specimen was carefully studied and revealed the following facts: The whole region of the hypophysis, both the cavernous sinuses and the anterior part of the clivus were invaded and occupied by a firm, light gray tumor, which was covered by dura (fig. 1). The dura had grown together with the tumor, and in places was thin, but at no point had it been penetrated. The tips of both petrous pyramids (fig. 1, *8*) had been destroyed by the tumor. The vertebral arteries were normal; the basilar and the right cerebellar arteries were likewise normal. The right trigeminal nerve perforated the dura at the typical place and divided into

three branches, situated lateral to the neoplasm. On the left side, the third branch went through the foramen ovale in the typical way (fig. 1, *7*), but the second branch had grown together in places with the tumor, and the first branch was completely embedded in it. The oculomotor and the trochlear nerves on the right side (fig. 1, *3* and *7*) also entered the tumor and were embedded in the mass, whereas the left oculomotor and trochlear nerves (fig. 1, *2*) were uninvolved and entered the superior orbital fissure. The optic nerves (fig. 1, *1*) and the ophthalmic arteries were normal. The hypophysis was embedded in the tumor, but the hypophyseal stem (fig. 1, *5*) was visible and free. The abducens nerve could not be identified on either side of the specimen with any degree of certainty. The internal auditory meatuses on both sides (fig. 1, *6*) were normal, as were the seventh, eighth, ninth, tenth and eleventh cranial nerves.

Fig. 1.—View at the base of the skull, from within the cavity of the skull; *1*, optic nerves; *2*, oculomotor and trochlear nerves on the left side; *3*, oculomotor nerve on the right side; *4*, planum sphenoidale; *5*, stem of the hypophysis; *6*, internal auditory meatus of the petrous bones; *7*, trochlear nerves; *8*, tips of the petrous bones; *9*, trigeminal nerve on the right side; *10*, abducens nerves; *11*, trigeminal nerve on the left side; *12*, cerebellar artery; *13*, area of the cavernous sinus; *14*, margin of the tumor.

The floor of the sphenoid sinus on both sides was infiltrated by the tumor. The right sphenoid sinus was almost entirely filled with tumor, while the left was only partially occupied. The roof of the epipharynx (fig. 2) was completely filled with necrotic and hemorrhagic tumor tissue. A large recent blood clot was adherent to the left wall of the nasopharynx, and the mucous membrane was entirely destroyed, whereas the right side was thickened and only partially necrotic.

On removal of the tumor in the epipharynx, marked erosion of the bony roof of the structure was noted. The tumor had extended far into the tips of the petrous

pyramids, which form the lateral portion of the roof, destroying them. The left internal carotid artery (fig. 2, *3*), just below its entrance into the temporal bone, showed a circumscribed aneurysmal dilatation (fig. 2, *2*), which had perforated on its medial aspect into the tumor (fig. 2, *1*). This was the source of the final fatal nasopharyngeal hemorrhage. Although the base of the skull was extensively involved by tumor, the jugular bulb and internal jugular vein were unaffected. The aneurysmal dilatation of the left internal carotid artery was observed to be compressing the glossopharyngeal nerve as it emerged from the skull.

The retropharyngeal gland and the deep cervical glands on the left side were enlarged and firm and, like the internal pterygoid muscle, contained tumor tissue. There were also several arteriosclerotic plaques in the external carotid artery. After

Fig. 2.—View at the roof of the epipharynx. A probe (*1*) is seen within the internal carotid artery on the left side indicating the perforation in the aneurysmal sac (*2*); *3* indicates the internal carotid artery.

examination of the intact base of the skull, the tumor was carefully removed and the specimen dissected; the findings were as follows (fig. 3): The sella turcica (fig. 3, *2*) was completely destroyed and the hypophysis mechanically displaced but not invaded, as was confirmed microscopically. The tumor covered both of the posterior clinoid processes and invaded the clivus Blumenbachi (fig. 3, *3*). Though the surface of this clivus appeared to be roughened, it was not invaded. The tumor extended from both sides of the sella turcica into the cavernous sinus and surrounded both carotid arteries, compressing the latter moderately.

The tumor destroyed the petro-occipital suture, the anterior foramen lacerum, the origin of the greater wing of the left sphenoid body and then extended into the cranial vault (fig. 3, *7*). The tumor also destroyed the left gasserian ganglion and all three branches of the left trigeminal nerve. The foramen ovale on the

left side (fig. 3, *6*) was normal except along its posterior border, where it was eroded. The foramen spinosum on the left was normal.

Thus, in this case extensive invasion by the tumor was observed, involving the body of the sphenoid bone (with a special predilection for the vascular marrow spaces), the sphenoid sinuses and the tips of the petrous pyramids, with destruction of the outer table of the base of the skull. Intracranial extension of the tumor occurred by way of the petro-occipital suture, the foramen lacerum anterius and the origin of the great wing of the sphenoid body. The tumor did not penetrate the dura, but in its extension grew together with it and thinned it by expansion. The growth of the tumor was almost entirely upward, although there was some lateral spread into the vault of the epipharynx. The tumor grew by eroding the bone and replacing it, except in the sphenoid sinus, where it pushed the mucosal lining ahead of it.

Fig. 3.—View at the base of the skull after the removal of the tumor; *1*, planum sphenoidale; *2*, area of the sella turcica; *3*, clivus Blumenbachi; *4*, tips of the petrous bones; *5*, superior angles of the petrous bones; *6*, foramen ovale on the left side; *7*, fissure in the base of the skull produced by invasion of the tumor.

The soft palate evidenced slight infiltration on the left side of the midline. On its nasal surface was a shallow ulcer with a necrotic base and irregular margins. Since this area of the soft palate came into contact with the tumor on the left side of the nasopharynx, it was regarded as another instance of implantation of carcinoma by contact from the nasopharynx to the soft palate. The microscopic section (fig. 4) revealed an area on the nasal surface of the soft palate devoid of epithelium and small groups of tumor cells near the surface. These were present in a small area near the surface. At one place in this area tumor cells were seen in what appeared to be a small lymph vessel (fig. 4 *d*). It is believed that this was an example of contact implantation carcinoma similar to that in the case described by Brunner,[10] because the drainage of lymph from the nesopharynx was not directed

10. Brunner, H.: Carcinoma of the Epipharynx: A Peculiar Case, Arch. Otolaryng. **29**:544 (March) 1939.

to this site and also because the carcinoma cells were observed in small groups solely in the ulcerated area and near the surface. Further study revealed only inflammatory changes in the surrounding submucosa and no evidence that the tumor cells had extended into this area by submucous spread. That there were tumor cells in a lymph vessel in this region might be explained by the fact that implantation cells had been taken into the lymphatics of the submucosa at the site of the ulcerated area. It is quite possible that distant metastases may have come from this area rather than from the original tumor, as distant metastases occur infrequently with

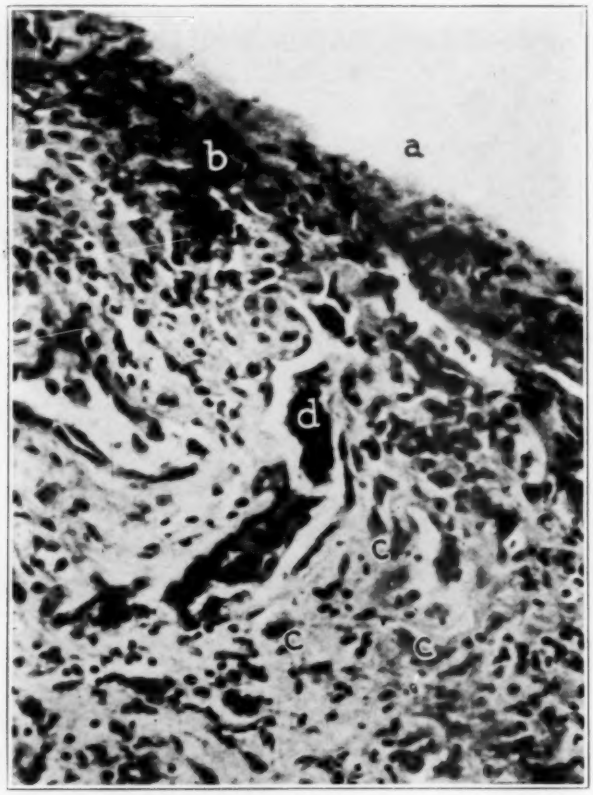

Fig. 4.—Section through the soft palate; *a*, nasal surface of the soft palate without the epithelial layer; *b*, subepithelial infiltration; *c*, tumor cells within the connective tissue; *d*, tumor cells within a lymph vessel.

carcinoma of the nasopharynx. The tongue and esophagus showed no evidence of gross pathologic change. In the larynx, the left ventricle of Morgagni consisted of a large opening with a considerable recess upward. The right ventricle was much smaller and narrower.

COMMENT

Although classic symptoms and findings of nasopharyngeal carcinoma, consisting of headaches, nasal hemorrhage, tinnitus, conduction deafness, paralysis of the abducens nerve, diplopia, neuralgia and later

swelling of the cervical glands, were present in the patient who is made the subject of this study, the diagnosis was not made until late, because a tumor was not seen projecting into the nasopharynx. The neurologic diagnoses were vague. Roentgenograms were reported as showing no definite pathologic change, in spite of marked destruction of bone and intracranial invasion later observed at autopsy.

The description of the pathologic process emphasizes the practically hopeless character of the disease when neurologic symptoms and findings are present, even if it is diagnosed correctly. The description of the pathologic process, furthermore, shows the great importance of an exact autopsy in such cases; conclusions even from an exact clinical observation may so often be altered by the results of autopsy. Such was the experience in this case. From pure clinical observation, the tumor apparently originated from the right nasopharyngeal wall and extended into the skull and probably progressed intracranially by way of both superior orbital fissures into the orbits. The autopsy, on the contrary, indicated that the tumor originated in the left nasopharyngeal wall, broke through into the skull by way of the petro-occipital suture and the anterior foramen lacerum at the point of origin of the greater wing of the sphenoid body and, always situated extradurally, invaded the orbits. Contrary to the observations of Brunner,[10] in this case the inner table was perforated. Carcinoma cells were implanted from the tumor in the nasopharynx to an ulcer in the soft palate by contact. This is in accordance with the observation of Brunner. There was also a metastasis to the liver.

It is noteworthy that in our case obvious clinical symptoms referable to the pituitary body were not present, although it was markedly compressed and displaced. A sidelight of interest in this case was the nystagmus obtained by vestibular stimulation in the right eye, which had complete paralysis of the abducens and oculomotor nerves, showing that nystagmus can be elicited with the superior oblique muscle alone.

The importance of this case report is that the study is presented from a complete clinical and pathologic point of view. We believe that knowledge, so far as early diagnosis, prognosis and treatment of this type of case is concerned, will be greatly facilitated by the publication of such combined observations rather than the presentation of large numbers of clinically observed cases.

Abstracts from Current Literature

Ear

THE VALUE OF SULFANILAMIDE IN OTOGENOUS INFECTIONS, WITH SPECIAL REFERENCE TO ITS MASKING EFFECT. JACOB L. MAYBAUM, EUGENE R. SNYDER and LESTER L. COLEMAN, J. A. M. A. **112**:2589 (June 24) 1939.

The authors give full credit to sulfanilamide but emphasize their opposition to its indiscriminate use. They state that its use has a tendency to obscure the course of an infection. For acute otitis media it should be used with caution, if at all. It should never be used for suppurative otitis media. They used the drug only for complications of otitic suppurations, such as meningitis, sinus thrombosis and abscess of the brain. Thus far scant attention has been paid to the masking effect, and little reference to this possibility is found in the literature.

Six interesting cases are given, which illustrate the points brought out in the article, which they conclude and summarize as follows:

"The use of sulfanilamide has resulted in a genuine advance in the treatment of certain otogenous infections.

"Exenteration of the otitic focus is of paramount importance.

"Indiscriminate use of the drug may obscure the diagnosis or result in a masked or latent clinical picture of mastoid involvement.

"Under sulfanilamide therapy, the usual clinical course of an otitic complication may be modified."

GORDON, Philadelphia.

SYMPTOMS AND COURSE OF MASTOIDITIS FOLLOWING OTITIS FROM SWIMMING. M. BUCHBAND, Monatschr. f. Ohrenh. **73**:332 (May) 1939.

A special type of surgical otitis, produced by infections acquired in swimming, is evaluated by the author. The infection can occur by way of the eustachian tube or by rupture of the drum by some external force, i. e., in diving or in water polo. In seven years, 20 such cases of surgical otitis were observed at the Neumann Otologic Clinic. In 16 of these the former and in 4 the latter path of infection was noted. All varieties of bacilli were found with this type of otitis, but the author cites an epidemic of 21 infections with the Friedländer bacillus. The course in all the cases in no way differed from that of other forms of surgical otitis, except that the death rate was double the normal, ruptured drums especially producing a more fulminant course. It may be reasoned that in these cases the infection meets no adequate resistance by the mucous membrane, because of its rapid onset.

LEDERER, Chicago.

INFLUENCE OF PARATHYROIDECTOMY ON THE HEARING IN CASES OF OTOSCLEROSIS. JUSTO M. ALORRSO and ALBERTO CHIARINO, Acta oto-laryng. **27**:123, 1939.

The authors present the effect of parathyroidectomy on the hearing in 19 cases of otosclerosis. After parathyroidectomy or even after ligature of branches of the left inferior thyroid artery they generally observed a more or less marked improvement in hearing. The hearing receded somewhat after the lapse of time but always remained at a higher level than before operation. The improvement of hearing was more marked for the spoken voice than for the whisper. In most of the patients the improvement was more marked on the side operated on. The results were in general better in the patients in whom the gland was extirpated than in those in whom branches of the inferior thyroid artery were ligated. The

best results were obtained in young subjects and in those in whom the otosclerosis had been present for only a few years. Their procedure does not contraindicate the subsequent performance of some sort of "fistulization" operation.

GROVE, Milwaukee.

TREATMENT OF PATIENTS WITH ACUTE SUPPURATIVE OTITIS MEDIA, ESPECIALLY WITH REGARD TO THE VALUE OF SYRINGING WITH ALCOHOL. VIGGO SCHMIDT, Acta oto-laryng. **27**:136, 1939.

After discussing the usual care of the ear after early paracentesis the author states that he uses hot compresses for twenty-four hours. He then begins to syringe the ear with a 33 per cent (by volume) solution of alcohol in a 10 to 20 cc. record syringe. This prevents maceration of the skin by pus or by water and by its shrinking action tends to keep open the perforation in the drum and also to open the tube, thereby promoting better drainage. The number of such syringings varies from three to six or even more per twenty-four hours, depending on the severity of the otitis. When syringing with alcohol is begun early after paracentesis pain from the alcohol itself is avoided.

Syringing with alcohol after paracentesis has reduced the number of mastoidectomies from 37.4 per cent to 13.5 per cent, and these figures were arrived at by covering four years in each instance.

Two hundred and eighteen patients so treated were reexamined a year or more after cessation of treatment. In 208 the hearing was normal; 210 had a healed membrana tympani. The response to the functional test was considered normal if the whisper could be heard at 20 meters. Only 2 patients had a moist middle ear. The author believes that all such patients should be hospitalized.

GROVE, Milwaukee.

SURGICAL TREATMENT OF VERTIGO BY OPENING THE EXTERNAL SEMICIRCULAR CANAL AND INJECTING ALCOHOL. W. W. MOLLISON, Acta oto-laryng. **27**: 222, 1939.

Mollison reports 50 cases of intractable vertigo, in most of which the condition was classified as Ménière's syndrome, in which he exposed the external semicircular canal by simple mastoidectomy. After exposure of the prominence of the external semicircular canal, it was opened by a gouge near its anterior end, and a few minims of absolute alcohol were injected into the vestibule. Only in 2 cases was fluid seen to leak from the opened canal. In 1 case there was transient paralysis of the facial nerve, which cleared up in three weeks.

The attacks of vertigo had continued over one to nine years and were so severe as to incapacitate the patients. Operation was performed only when the hearing of the opposite ear was relatively good and that of the affected ear bad.

Some of the patients had tried various medical treatments and all had taken phenobarbital for several weeks without relief. Some had had a limited water intake and a salt-free diet. Five had had previous mastoidectomies. No patient died as the immediate result of the operation nor did any show signs of infection of the labyrinth. One patient died of nephritis some weeks after the operation.

Seventy-four per cent of the patients described themselves as cured; 10 per cent were not benefited, and the remaining 16 per cent either were improved though not cured or could not be traced.

GROVE, Milwaukee.

Pharynx

OSTEOMA AT THE BASE OF THE TONGUE. TETSUSABURO HITA and KEN OGITA, Oto-rhino-laryng. **11**:1016 (Nov.) 1938.

Review of the literature indicates that osteoma of the base of the tongue is exceedingly rare. In 1913 Monserratt of France reported a case of osteoma. In 1926 Hirsch reported a case of an osteoma the size of a cherry seed in a woman

26 years of age. Hita and Ogita report a case of osteoma in a man 23 years of age. The patient's chief complaint was dysphagia of four months' duration. The tumor was smooth on palpation and located slightly to the right of the median line. The growth was excised with the patient under local anesthesia. It measured 6 by 5 by 4 mm. Hirsh was uncertain whether it was due to embryonic misplacement or metaplasia of the thyroid gland. Histologically Hajek was unable to identify thyroid tissue in the tumor. Glos does not believe in metaplasia of thyroid tissue but advances a theory that osteoma may be found in this region for the same reason that faucial and lingual tonsils occasionally contain bony tissue.

HARA, Los Angeles.

RHINOPHARYNGITIS MUTILANS ON THE ISLAND OF PALAU. MASAO SEKINE and MASAO YOKOO, Oto-rhino-laryng. **12**:3 (Jan.) 1939.

The writers report 12 cases of gangosa observed by them on the island of Palau. Travelers in the tropics have long noted natives whose noses have been destroyed and who present characteristic scars on their faces. Some authors attributed the condition to tropical frambesia as a primary cause, and others called it rhinopharyngitis mutilans, or gangosa.

In 1928 Schöbel was able to produce in a Philippine monkey clinical gangosa by introduction of Treponema pertenue. The writers report 5 cases in which the actual cause was frambesia tropica. In typical cases the face is greatly distorted by the formation of scars; the external nose, septum and turbinates undergo varying grades of destruction, leaving the passage narrow as the result of contracture. The palpebral fissures are narrowed, and movements of the eyes are impaired. In advanced conditions the hard palate suffers necrosis; teeth fall out, and the soft palate adheres to the posterior and lateral pharyngeal wall. The motility of the tongue is limited, and a characteristic nasal tone of speech develops, the description of which is the Spanish word *gangosa*. In the remaining cases the condition had been called by the Annamese term *ngardik*, the native name for framboesia tropica during the second stage of its development. In the earlier stage the disease is manifest by narrowing of the nasal passages and gradual softening of the gums and nasal septum. Later this process is followed by complete collapse of the external nose and scarring of the skin and muscles on the face. The disease is most often met after middle life, though one of the cases reported was that of a boy 14 years old. The entire island has a native population of 6,022 according to the census of 1934. Besides the 12 cases 2 additional cases of gangosa were known to the writers. The paper contains six photographs of the patients and one table.

HARA, Los Angeles.

A CASE OF FIBROMA OF THE HARD PALATE. TOSHIO KABASHIMA, Oto-rhino-laryng. **12**:34 (Jan.) 1939.

Tumor of the hard palate, particularly of a benign type, is rare. Kabashima reports a case of fibroma of the hard palate in a woman 45 years of age who had noticed the growth for five or six years. Recently she had experienced gradual alteration of the voice, and she requested an examination. The tumor was pedunculated and sprang from the center of the hard palate, with a stalk 5 mm. in length. It was removed with a hot snare. It measured 3.3 by 2.3 by 1.2 cm. The surface was smooth, thickest at the center and shaped like a pearl button. Review of the literature regarding benign tumors of the palate in Japan led the author to the conclusion that nearly all kinds of neoplasm arise from the palate, the highest incidence being that of endothelioma. Next in incidence is mixed tumor. Osteoma and myxoma are not reported. Benign tumors of the palate are more common among females, occur more often in the soft than in the hard palate and are most frequently encountered during the fifth and sixth decades.

HARA, Los Angeles.

Nose

EXTERNAL OPERATIONS ON THE FRONTAL AND ETHMOIDAL SINUSES. NORMAN PATTERSON, J. Laryng. & Otol. **54**:235 (May) 1939.

Operations on the nose are to be avoided when possible, especially as patients who would otherwise make a good recovery are sometimes prevented from doing so by an operative procedure.

After considerable popularity, external operations on the sinuses fell into disrepute for some years because of the frequency of complications. Largely through the efforts of Walter Howarth, the external operation has regained its favor, and attention to details has reduced the incidence of complications. The external operation is not more dangerous than the intranasal, and the scarring, when the incision is properly performed, is negligible.

The frontal and ethmoid areas are so closely associated that the former is almost never opened without also entering some of the cells of the latter. It is possible, on the other hand, to examine the frontal sinus after directing attention to a diseased ethmoid.

In order to secure the most satisfactory cosmetic result compatible with adequate exposure of the diseased areas there are only two regions where the incisions should be made; one is in the neighborhood of the supraorbital ridge; the other is in the cheek. In some cases both incisions, separated by a considerable interval, are employed. The "frontal" incision passes along the supraorbital ridge and is at the center of or just below the hairy eyebrow. The hair should not be shaved, and the incision should not be carried beyond the hairy area. Through this incision all manipulations on the sinus can be carried out, and moreover, the frontonasal region can be explored or entered. The choice between obliteration of the sinus or simple drainage depends on the extent of the pathologic condition encountered.

The "ethmoid" incision begins about ¼ inch (0.6 cm.) below the level of the inner canthus, and it follows the natural sulcus, passing downward and outward into the cheek. It is about an inch (2.5 cm.) in length. The periosteum over the ethmoid area is elevated, and the cells are attacked from before backward. Tearing of the upper end of the incision is prevented by a stitch in the angle of the wound. After all the ethmoid cells are removed the sphenoid sinus can be inspected. By this incision the antrum can be easily entered from the upper part of its nasal wall. If necessary, a large nasoantral opening can be made entirely above the inferior turbinate. Gravitational drainage, in the recumbent position, will occur from such an opening, and if there are any active cilia, they will be effective in providing necessary drainage.

LE JEUNE and BAYON, New Orleans.

OSTEOMYELITIS OF THE SUPERIOR MAXILLA FOLLOWING INTRANASAL OPERATION. SHOJI TAKAMURA, Oto-rhino-laryng. **12**:14 (Jan.) 1939.

Osteomyelitis of the superior maxilla is sometimes met in children but rarely in adults except when of dental origin. Takamura reports the case of a 47 year old fisherman in whom intranasal resection was made for chronic infection of the righ maxilla. The evening of the operation swelling over the face, septic temperature, pain and foul discharge from the right nostril developed. Later an abscess developed over the right inner canthus. This was evacuated, but a fistulous tract persisted. The right nostril continued to be filled with a foul discharge. The floor of the nose showed sequestrums. The bony portion of the septum and the lateral wall of the right nostril showed a large perforation. Two months after the operation perforation of the hard and soft palate developed. The right maxilla had undergone complete necrosis, and the sinal cavity was covered with grayish ulcerative membrane. The exciting organism was the staphylococcus. Treatment consisted of both local cleansing and general systemic supportive measures. After an apparent improvement the patient became gradually weakened, and about fifty

days after the onset of the disease hemorrhage from the ulcerated area and death from circulatory failure occurred. Takamura emphasizes the importance of meticulous postoperative care in order to prevent such a complication.

HARA, Los Angeles.

RELATION OF CHRONIC BRONCHITIS AND PARANASAL SINUSITIS. SHIRO NAKAMURA, Ztschr. f. oto-rhino-laryng. **45**:5 (May) 1939.

During two years (1935 and 1936) the author observed at Keio University Hospital 15 cases of chronic bronchitis associated with paranasal sinusitis. Six patients were in the third and five in the fourth decade; 11 were males, and 4 females. Eighty-seven per cent of them complained of coughing. Others complained of nasal discharge, postnasal drip, nasal obstruction and impairment of the sense of smell. Because of cough and expectoration almost all the patients had consulted internists before being referred to the department of otolaryngology. Nasal examination revealed that 12 had chronic suppurative, and only 3 nonsuppurative, sinusitis. Eleven had chronic bronchitis and 3 bronchial asthma. Bronchiectasis developed in 1. Ten were asthenic, 1 phlegmatic and 4 more or less muscular. The author states that the condition was bilateral in nearly all. Physically the patients were highly nervous. He reviews various theories as to the underlying causes of bronchitis and sinusitis, such as deficiency of vitamins, allergic predisposition and low sugar content of the blood, but he advances a theory that bronchitis as a complication of sinal infection is most frequently observed in the highly neurotic.

HARA, Los Angeles.

Miscellaneous

ALLERGY IN INFANTS: SIGNIFICANCE OF FIRST ALLERGIC MANIFESTATIONS. N. W. CLEIN, Northwest Med. **38**:9 (Jan.) 1939.

A series of 100 allergic and 100 nonallergic infants was studied from birth to the age of 4 or 5 years. The feedings, examinations and observations were uniform in all cases.

Of the allergic infants, 71 per cent had allergic parents; of the nonallergic, 12 per cent.

Of the allergic infants, 78 per cent exhibited some allergic symptoms before the fourth month of age, 91 per cent by the end of the first year and 100 per cent by the end of the second year, as follows: rash (eczema), occurring chiefly on the face (85 cases); vomiting (pylorospasm) (24 cases); colic (abdominal pain, gas or diarrhea) (16 cases). Frequently one or more of these symptoms were present at the same time.

Egg yolk should not be added to the diet of allergic infants too early, as many are sensitized through the mothers' milk or inherit the sensitivity.

Taking of a family history, study of the present allergy, elimination and testing are often necessary for a proper solution.

ROBERTSON, Omaha. [AM. J. DIS. CHILD.]

STAPHYLOCOCCUS SEPTICEMIA. P. F. STOOKEY and L. A. SCARPELLINO, South. M. J. **32**:173 (Feb.) 1939.

In a compilation of the records on 117 cases of staphylococcic septicemia from various hospitals the authors found the gross mortality to be 91.4 per cent. Among 28 children less than 18 months of age the mortality was 78 per cent, and among those over 18 months of age it was 94.1 per cent. In 17 patients treated with staphylococcus antitoxin the mortality was 53 per cent.

An early diagnosis of staphylococcic septicemia should be made and treatment carried on with adequate amounts of antitoxin to prevent irreparable damage of tissues.

Although the mortality was markedly lowered by the early administration of staphylococcus antitoxin, the ideal biologic procedure has not been attained.

SCHLUTZ, Chicago. [AM. J. DIS. CHILD.]

A STUDY OF BONE REGENERATION. GUSTAV LEVANDER, Surg., Gynec. & Obst. **67:705, 1938.**

The specific osteoblastic theory holds that regeneration of bone may take place from preexisting cells belonging to the different layers; the metaplastic theory maintains that bone may be formed as a result of a process of transformation of nonspecific connective tissue. An essential part of the first theory concerns the predominant role of periosteum. Experimental evidence, however, has shown that it is only the periosteum of the growing skeleton that has the power to stimulate the formation of bone. Also it is known that when fully differentiated hard bone tissue is grafted, new bone is always formed. In a series of rabbits Levander first studied the mode of origin of new bone after transplanting into soft parts hard bone tissue stripped of periosteum. New bone formed from the mesenchymal tissue in the areas surrounding the graft. In his opinion, the specific stimulus necessary for the formation of new bone is brought to the mesenchyme in the form of a substance liberated from the graft and conveyed with the lymph into the surrounding areas. In a second series of experiments alcoholic extract of bone tissue were injected into soft tissues of the abdominal wall as far as possible from any preexisting bone. Cartilage or bone was formed at the seat of injection in 22 per cent of the rabbits. Alcohol alone or alcoholic extracts of other tissues gave negative results in a control series. On the basis of these experiments it is felt that bone regeneration takes place as a result of activation of the nonspecific mesenchymal tissue by some specific bone-forming substance. This supports the heteroplastic theory of the formation of bone and conforms with the views of Spemann with regard to embryonic development.

WARREN C. HUNTER. [ARCH. PATH.]

SOME OBSERVATIONS ON HEADACHE. D. W. C. NORTHFIELD, Brain **61:133, 1938.**

Headache was the initial symptom in 36 of 100 unselected cases of verified tumor of the brain. It was not the first symptom in 9 cases of acoustic neurinoma, but it was the initial symptom in 10 of 12 cases of tumor of the posterior fossa. The incidence for gliomas of the cerebral hemispheres was similar to that for cerebral meningiomas. Headache was the initial symptom in more cases of tumor of the temporal than of the frontal lobe. Headache is likely to precede other symptoms in cases in which the tumor, by reason of its location, tends to impede the flow of cerebrospinal fluid through the ventricular system. Of 121 cases, excluding those of pituitary adenoma, frontal headache was absent in only 5 per cent, as compared with 25 per cent of those in which the tumor was infratentorial. Pain in the neck was present in 22 per cent of cases of supratentorial tumor and in 49 per cent of cases of infratentorial tumor. The headache was entirely or predominantly unilateral in 42 cases and did not shift from side to side. In 6 of these cases the tumor was situated approximately in the middle line, but in only 4 cases was the tumor on the side opposite that of the headache.

It is commonly accepted that the headache produced by an intracranial tumor is due to increase in intracranial tension. Of a series of 102 cases of verified tumor of the brain, the pressure exceeded the normal in 63; in 39 it was less than 200 mm. of fluid, and in 16 it was below 100 mm. In many cases in which the pressure was normal or subnormal there was severe headache. In many cases headache is absent although the intracranial pressure is high. These cases demonstrate that the absolute value of the intracranial pressure bears no relation to the presence or absence of headache. Although headache is clearly related to diseases which produce an increase in intracranial pressure, the high pressure in itself is probably not the cause of the pain.

Certain conditions other than intracranial tumor suggest the possibility that headache may be vascular in origin. Patients suffering from an intracranial aneurysm often complain of recurrent headache, which commonly affects one side of the forehead more than the other. This suggests that the gradual stretching of the wall of the artery and the final rupture of the aneurysm may set up a train of afferent stimuli which is interpreted as headache.

During operative procedures, observations were made to determine the relative sensitiveness of various tissues. In the majority of cases the traction necessary to produce pain on stimulation of the dura mater was considerable. The tentorium seemed to be more sensitive than other areas of the dura mater, except that around the middle meningeal arteries; the dura mater lining the vault was the least sensitive. Pressure on or displacement of the internal carotid artery in the vicinity of the anterior clinoid process resulted in pain in or near the homolateral eye in 3 cases, pain in the homolateral temple in 1 case and no sensation in 1 case. In no case did sensation of any kind result from pressure on the pia mater, the brain tissue or the walls of the ventricles. The cortical veins and arteries and the choroid plexus were insensitive to clamping and to endothermy.

The effect of raising the intraventricular pressure was noted in 4 cases. Warm Ringer's solution was injected quickly into the ventricles in the course of ventriculographic examination, before the removal of any cerebrospinal fluid. Headache resulted in 2 cases. In 3 cases of tumor high in the spinal cord Ringer's solution was injected into the cisterna magna. In 1 case, after the injection of only a few cubic centimeters of fluid, the patient complained of severe frontal headache. Twenty cubic centimeters of Ringer's solution injected under the dura mater in the course of cerebellar exploration, probably into the subdural space, has resulted in homolateral occipital headache, which gradually disappeared as the fluid escaped. Compression of the jugular veins for two minutes induced headache in 6 of 9 cases, and in 4 of these the resulting intracranial pressure was not above 200 mm. of water.

In many cases headache was produced by suddenly reducing the intracranial pressure. In 5 cases in which the lateral ventricles were tapped, the sudden escape of fluid was attended by considerable pain.

Headache does not occur during the ventriculographic procedure, whereas it is intense during encephalographic examination.

From study of clinical material and experimental observations it appears that the sudden increase or decrease of pressure, previously normal or abnormal, readily causes headache. Rapid reduction of pressure is perhaps the more conspicuously successful method. The conclusion may be drawn that it is the sudden variation in the intracranial pressure, and not the steady pressure itself, which induces experimental headache.

Histamine phosphate was injected into 14 patients suffering from tumor of the brain, and in 11 cases the headache which occurred was similar in quality and distribution to that produced by the tumor. During the course of cerebral arteriographic examination histamine phosphate was injected into the common carotid artery in 13 patients, resulting in homolateral headache in 6 cases and bilateral headache in the others, the headache being more severe on the homolateral side. The injection of histamine in the internal carotid artery resulted in homolateral headache in all of 5 cases. In the same 5 cases, injections into the external carotid artery did not produce headache. Division of the middle meningeal vessels did not prevent bilateral headache after the intravenous injection of histamine.

The only intracranial tissue other than the dura mater which is sensitive to suitable stimuli is apparently the cerebral blood vessels. It is Northfield's belief that the headache associated with intracranial tumor is due to disturbance of the tension of the walls of the cerebral vessels. It is this disturbance which provides the adequate stimulus for the excitation of pain.

SALL, Philadelphia. [ARCH. NEUROL. & PSYCHIAT.]

OTITIC ZONA, ASSOCIATED WITH ZONA OF THE TRIGEMINAL, GLOSSOPHARYNGEAL AND SUPERIOR LARYNGEAL NERVES. BALDENWECK and DEGORCE, Rev. d'oto-neuro-opht. **17**:29 (Jan.) 1939.

Baldenweck and Degorce report the observation of a case of otitic zona, with involvement of the right fifth, ninth and tenth cranial nerves. Both the cochlear and the vestibular division of the eighth nerve were involved. The eruption appeared on both sides of the epiglottis. The question is raised whether the upper cervical plexus was involved. Despite the occurrence of the eruption also in the submaxillary and retroauricular regions, it is suggested that this may have been due to involvement of the cervical branch of the seventh and of the recurrent sensory branches of the fifth nerve, respectively. Furthermore, the opinion is expressed that it is more logical to believe that the seat of the lesion was in the bulbar nuclei rather than that a number of ganglions were involved. Support for this opinion is supplied by the facts that the paralysis was limited to the upper branch of the facial nerve and that the left cochlear and vestibular nerves also were affected to some degree.

DENNIS, San Diego, Calif. [ARCH. NEUROL. & PSYCHIAT.]

HISTOLOGIC CHANGES IN THE EAR, NASOPHARYNX, LARYNX AND TRACHEA PRODUCED BY SPLENECTOMY. G. ZANZUCCHI, Arch. ital. di otol. **51**:38 (Jan.) 1939.

Numerous investigations of the effect of splenectomy on other organs have been done, and it has been shown that removal of the spleen produces in the organism no lasting damage, because other organs assume a vicarious function. The immediate effects are reactions in the blood and in the organism as a whole: the appearance of immature red cells in the peripheral circulation, leukocytosis and congestion of the liver, kidneys, bone marrow and lungs. Busacca observed in splenectomized dogs slight hyperplasia of the tonsillar tissue two months after the operation followed by a marked involution at the fifth month. Zanzucchi removed the spleen in 22 adult rabbits, killed them in groups of 5 or 6 at intervals of one, two, four and eight weeks after the operation and then investigated the histologic changes in the ear and upper air passages. In the noses of all of the first and second groups congestion and hemorrhages were found in the submucosa; in half of them similar changes were found in the pharynx, larynx and trachea and in their associated lymph glands; in the medulla of the bone of the internal ear congestion was noted in half the cases. In the third group the changes were less marked, and in the fourth group the changes were even less or were absent. The observations agree with those of investigators concerned with alterations in other organs and tissues.

DENNIS, San Diego, Calif.

ROLLING MOVEMENTS IN PIGEONS. E. HUIZINGA, Arch. f. d. ges. Physiol. **240**: 713, 1938.

Huizinga studied the rotation of the head appearing in pigeons several days after unilateral labyrinthectomy. If one labyrinth is extirpated first and the second labyrinth after several months, the symptoms of deficiency are much more marked than after the first labyrinthectomy; rotation of the head appears within twelve hours. These phenomena suggest the importance of the compensatory process in the vestibular nuclei in the genesis of the rotation. Rolling movements appeared in 1 animal. They are due to the marked rotation of the head, which finds no support.

SPIEGEL, Philadelphia. [ARCH. NEUROL. & PSYCHIAT.]

ACOUSTIC HALLUCINATIONS AS THE PRODROMAL SYMPTOM OF SCHIZOPHRENIA. G. J. VLAVIANOS, Monatschr. f. Ohrenh. **73**:68 (Jan.) 1939.

A man 44 years old suddenly noticed tinnitus in both ears and heard voices talking to him. Pathologic changes in the ear were not observed. The author believes that the tinnitus was not produced by an otic disease but may be the first symptom of schizophrenia.

LEDERER, Chicago.

TREATMENT WITH VITAMINS IN OTOLARYNGOLOGY. A. EROEDI, Monatschr. f. Ohrenh. **73**:95 (Feb.) 1939.

The efficiency of vitamins in otolaryngologic practice is briefly summarized. The writer stresses the importance of vitamin C, which is supposedly a specific remedy for all acute ulcerations within the mouth, particularly Plaut-Vincent's angina.

LEDERER, Chicago.

SALIVARY CALCULI AND SIMULTANEOUS INFLAMMATION OF THE TESTICLES. A. BARUCHOFF, Monatschr. f. Ohrenh. **73**:117 (Feb.) 1939.

The writer describes a case of a calculus within Wharton's duct, combined with an inflammation of the testicle on the same side. After spontaneous expulsion of the stone the inflammation of the testicle subsided.

LEDERER, Chicago.

HEMORRHAGES OF THE ESOPHAGUS PRODUCED BY FOREIGN BODIES OFFERING DIAGNOSTIC DIFFICULTIES. J. DANIELEWICZ, Monatschr. f. Ohrenh. **73**:121 (Feb.) 1939.

A woman swallowed a piece of glass, after which she experienced difficulty in swallowing and expectoration of blood. The foreign body was found within the isthmus of the esophagus and was removed. Recovery followed. In another instance a woman had swallowed a bone six weeks before coming under observation. During this time she had pain on swallowing, general malaise, melena and bloody expectoration. Roentgen findings were entirely negative. During her stay in the hospital two hemorrhages occurred. A subsequent roentgen examination revealed stasis of the barium paste at the level of the jugular vein. A bone was found at the isthmus of the esophagus by means of the esophagoscope and was removed. Recovery was uneventful.

LEDERER, Chicago.

THE IMPORTANCE OF THE PSYCHOLOGIC FUNCTION IN THE SYNDROME OF STAMMERING. L. PATZAY-LIEBERMANN, Pract. oto-rhino-laryng. **2**:54 (Feb.) 1939.

The author discusses the results of an investigation into psychologic effects on stammering. He believes that single elements of the syndrome can be understood only if considered from a "global" standpoint. He analyzes the concomitant movements of stammerers from the standpoint of their expressional content and concludes that in the moment of stammering the entire function of expression is submitted to a dispersion and those parts of the expression which cannot be uttered verbally appear as mimic (motor) symptoms. He further analyzes the psychologic conditions which favor the occurrence of stammering.

PERSKY, Philadelphia.

TREATMENT OF CONGENITAL ATRESIA OF THE NASOLACRIMAL DUCT. S. LARSSON, Acta ophth. **16**:271, 1938.

The nasolacrimal duct may be longer than the bony canal containing it. In such cases it continues below the orifice of the bony canal, its medial wall being covered by nasal mucous membrane. Its normal opening may form a window on the medial wall above its end. For this reason there are certain patients with congenital atresia of the lacrimal duct who cannot be successfully treated by probing, for the probe passes to the floor of the nasal fossa without producing a perforation medially. In 2 such instances the author has removed the tip of the inferior turbinate and incised the mucosa against a probe which had previously been introduced in the usual way.

PERKINS, New York. [ARCH. OPHTH.]

Society Transactions

MASSACHUSETTS EYE AND EAR INFIRMARY

Cases Selected from the Regular Weekly Clinical Meetings from January 1938 to January 1939

EDITED BY R. L. GOODALE, M.D., BOSTON

Toti-Mosher Operation. DR. G. H. POIRIER.

An automobile salesman came to me with a history of hospitalization for eight weeks with a fractured skull and an infected scalp incurred in an airplane accident. On his discharge from the hospital he had slight lacrimation of the left eye. He consulted an ophthalmologist, who probed the lacrimal apparatus, causing him a good deal of pain. During the following year he consulted a dozen or more ophthalmologists. He had a "colored fluid" injected through the duct, most of which came into the nose and throat.

On examination he had the typical membrane over the tear sac. Pressure caused pus to exude from the upper and the lower canaliculus. Roentgenograms made after injection of iodized poppyseed oil into the sac showed that the oil went through the canaliculus into the sac and then onto the floor of the antrum. That is, it went through the various passages made by the operators. Normally it should have gone through the nasolacrimal duct toward the floor of the nose.

A Toti-Mosher operation was done six weeks before the date of this report. Since then the patient has been entirely free from pus and tearing and has no symptoms referable to the antrum. The point which I wish to bring out in connection with this case is that the injection of iodized poppyseed oil and the roentgenograms were a great help in tracing the symptoms. I think most of the ophthalmologists were led astray by the colored fluid going into the nose and throat. The antrum was not infected.

DISCUSSION

DR. W. B. HOOVER: I think this is a unique case. I do not think that anybody has demonstrated a connection with the antrum before. If the stricture is near the canaliculus of the sac, does Dr. Poirier do a Toti-Mosher operation and then put in a device between the sac and the canaliculus?

DR. G. H. POIRIER: There is a good reason for doing a Toti-Mosher operation if there is stenosis of the lacrimal duct. When dealing with a partly or completely blocked canaliculus, one can do a Toti-Mosher operation, and in addition to that one can then slit the canaliculus as far as its juncture with the anterior wall of the sac, making one big hole which will do the work without any permanent style or other instrument. Possibly one might have to dilate that large slit a few times for a few weeks after the operation, but it works well.

DR. H. P. MOSHER: This was, evidently, a case of fracture.

DR. G. H. POIRIER: There is no question in my mind that probing is often a cause of dacrocystitis.

Bilateral Aerocele and Abscess of the Neck. DR. C. T. PORTER and DR. W. J. WHERRY, with Roentgenologic Report by Dr. A. S. MACMILLAN.

DR. W. J. WHERRY: A 58 year old man was first seen in the outpatient department in September 1937, complaining of hoarseness for the previous four

months. The left vocal cord could not be seen by indirect laryngoscopic examination, because of a cauliflower-like, reddish gray, lobulated tumor in the left ventricular region. There was no limitation of motion of the right cord. The differential diagnosis at this time was syphilis, malignant disease or tuberculosis. The patient was admitted to the hospital for biopsy. The report was chronic inflammation. Roentgenograms of the chest showed no pathologic change. The Hinton test indicated the presence of syphilis. During the next six months the size of the tumor remained about the same, and on two occasions biopsy was again performed to determine the question of malignant disease, syphilis or tuberculosis. In spite of persistent negative reports, the staff of the tumor clinic felt that the growth must be malignant and instituted roentgen treatment.

Nine months after the first visit, roentgenograms showed air spaces on each side of the hypopharynx, which continued to increase, to an extent sufficient to make respiration difficult. Because of the bilateral aeroceles and an anterior swelling of the neck due to perichondritis of the thyroid cartilage resulting from the roentgen treatment, tracheotomy was necessary.

Three months later, the anterior swelling of the neck had markedly increased and had become fluctuant. Dr. Poirier made a midline incision in the neck extending from the hyoid bone to the tracheotomy wound and obtained pus. The exploring finger could be passed easily on the left side of the larynx to the esophagus and could touch the cervical vertebra. On the right side the finger could not pass deeper than the larynx. A rubber drain was inserted for drainage.

The patient returned to the hospital three months later with a persistent draining fistula in the anterior part of the neck.

Two days ago, a midline incision was again made, and the exploring finger again passed on the left side as far as the vertebra. There was also a fistulous tract between the hypopharynx and the neck. No pus was encountered at this time. Roentgenograms have been taken from time to time to observe the progress.

DR. A. S. MACMILLAN: The first roentgenogram of the neck taken in this hospital, about one year after the patient first came in, shows the two large air spaces on either side of the spine. Then an abscess developed and one can see the marked increase in density in the pretracheal and laryngeal region. That abscess was drained, and the large air cells seemed to be much larger than they were before. One can see where a tracheotomy has been done. Now today, the neck shows marked swelling in front of the larynx.

DR. W. J. WHERRY: Dr. A. O. Freedman, of Montreal, gave a plausible explanation of this condition (Diseases of the Ventricle of Morgagni, with Special Reference to Pyocele of a Congenital Air Sac of the Ventricle, ARCH. OTOLARYNG. **28**:329 [Sept.] 1938). The sacculus, the lining of which contains numerous mucous and lymphoid glands, has been called the "oil can" of the larynx.

In the orang-utan and the howling monkey a large air sac connects with the ventricle of the larynx. The condition in the case just presented is comparable to this.

Dr. Freedman gave a plausible explanation for the persistent biopsy reports of chronic inflammation in this type of case. The aeroceles of the neck become infected, fill up and spill their contents continuously over the vocal cords, setting up a chronic inflammatory condition, which in time can produce a rather large lesion.

DISCUSSION

DR. H. P. MOSHER: What was the end result in the case?

DR. W. J. WHERRY: The patient was discharged from the hospital and has a normal voice.

DR. H. P. MOSHER: That certainly is a fortunate outcome.

Acute Pansinusitis on the Right, Meningitis, Septicemia, Extradural Abscess and Abscess of the Frontal Lobe. DR. C. T. PORTER, DR. P. MYSEL and DR. W. J. WHERRY, with Roentgenologic Reports by DR. A. S. MACMILLAN.

DR. W. J. WHERRY: A 21 year old boy was first seen in the emergency ward, on March 5, 1939. He had had an acute head cold for two weeks. Three days before admission he had had a severe shaking chill followed by persistent frontal headache on the right, nausea and vomiting.

On admission he was semicomatose, and he had partial paralysis of the left side of his face and left arm and complete paralysis of the left leg, stiffness of the neck and a positive Kernig sign. Lumbar puncture revealed an initial pressure of 220, with 453 cells, of which 86 per cent were polymorphonuclear leukocytes. The diagnosis was acute bilateral pansinusitis with extradural abscess and meningitis.

DR. A. S. MACMILLAN: A roentgenogram of the patient showed pansinusitis with both frontal sinuses, both ethmoid sinuses and both antrums full of pus. This is a picture of pansinusitis in the acute stage at the end of the first week. The lateral view, however, showed the sphenoid sinus perfectly clear and the floor of the frontal sinus dense. The frontal sinus, deep as it was and thin as the bone was around it, should have been as black as the sphenoid. The outline of a large diploetic vein could be made out in the skull. Such veins are regarded with suspicion, because when one observes pus in the frontal sinuses some of them run into the margin of the sinus itself.

DR. W. J. WHERRY: Because of the severity of the infection, the boy was operated on immediately. A bilateral radical frontal operation was done by Dr. Mysel. On removing the posterior wall of the right frontal sinus, an extradural abscess containing about 2 ounces (7.4 cc.) of pus was uncovered. The posterior wall of the left frontal sinus was uncovered, but there the dura was normal.

The next day the blood culture was reported to contain the beta hemolytic streptococcus, and sulfanilamide therapy was immediately instituted. Two days later, because of the herniation of the right frontal lobe through the bony defect, that lobe was probed for abscess of the brain. Nothing was found. For the next eleven days the patient's condition remained stationary. Because of the paralysis of the left side, the neurologic consultants felt that we were dealing with an abscess of the brain, high in the right frontal lobe.

On March 18 Dr. Mysel enlarged the bony defect over the right frontal lobe, removing the frontal bone upward for 3 inches (7.6 cm.). On probing on the upper portion of the exposed area a large abscess of the brain was tapped. A portion of the cortex over the abscess was removed and a Mosher wire basket inserted, the apex of which almost reached the bottom of the abscess. Since that time the patient has shown gradual improvement. The spinal fluid pressure has stayed around 260, and the cell count has dropped to 2. He does not have stiffness of the neck or paralysis, and, because of the consistently negative blood cultures, administration of sulfanilamide has been stopped.

On April 1 the right frontal lobe was probed inferiorly for a daughter abscess. Nothing was found.

For the last ten days the patient has shown definite improvement clinically, until yesterday, when he became irritable and complained of severe frontal headache. Lumbar puncture was done, and the initial pressure was 480, with 25 cells, of which 15 were polymorphonuclear leukocytes. At present he is listless, although easily aroused, and responds readily to questions.

DISCUSSION

DR. H. P. MOSHER: It certainly looks as if the treatment in this case is not over. The basket drain has to be kept clean and curetted. Of course, it can, as

in this case, close over at the end. It did close over, but in this case lies in line with all cases of abscess of the brain; after a long delay in healing, it looks like another case of abscess of the brain.

DR. GORDON BERRY: Two or 3 patients have been treated with sulfanilamide. The patients got better; the administration of sulfanilamide was stopped, and then the patients returned to the hospital.

DR. H. P. MOSHER: My associates and I have had the same experience. Patients sent out apparently better are coming back with meningitis.

Pansinusitis on the Right and Extradural Abscess. DR. H. G. TOBEY and DR. F. M. WATTLES, with a Roentgenologic Report by DR. A. S. MACMILLAN.

DR. F. M. WATTLES: A man aged 39 was admitted to the hospital on January 17 with a history of pain over the right temporal region with a stopped-up nose and a purulent discharge from the right side of the nose. He had been seen two weeks after the onset of the infection in the clinic, at which time a roentgenogram of the sinuses was taken. This was two weeks before his admission to the ward.

DR. A. S. MACMILLAN: Roentgenograms showed pansinusitis on the right side. The outline of the frontal sinus on the left side was not clear. There was pus in the right antrum and the right frontal sinus, too. Two small areas of increased density represented little exostoses on the inner table of the skull and on the posterior and anterior walls of the frontal sinus. But when the patient came in at the end of two weeks from the onset of the stuffed nose, he had definite pansinusitis on the right side.

DR. F. M. WATTLES: Previous to admission, he had had three washes of the antrum on the right side. The symptoms decreased markedly, but he complained of slight right frontal headache, and on January 17 it was thought that because he had not improved sufficiently he should be sent to the ward for further treatment.

DR. A. S. MACMILLAN: On the morning after the patient was admitted, because of what had happened since the first roentgenogram was taken, my associates and I looked carefully into the right frontal sinus again. The outline of that sinus was about the same as before, but there was an area of exostosis that was much clearer than before. Looking back, I think that there was decalcification of the posterior wall of the frontal sinus where the little osteoma was situated.

DR. F. M. WATTLES: One-half hour after the roentgenograms were taken the patient had a sudden generalized convulsion, which made him bite his tongue. He became cyanotic. He had four more convulsions of less severity in succession. The neurologic staff saw the patient and found no localizing signs at all, and they suggested either an epileptic attack of syphilis of the central nervous system or an abscess of the brain from the pansinusitis on the right.

The next morning the patient felt well, but because of the convulsion Dr. Tobey and Dr. Poirier decided to investigate the right frontal sinus. The leukocyte count on admission was 13,000, with 80 per cent polymorphonuclears. The chest was perfectly clear up to the day of operation.

On January 20, three days after admission, the patient was operated on by Dr. Poirier. At this time, with the patient under ether, a radical operation on the right antrum was done. There were thick creamy pus and necrotic bone in the antrum. Then an external orbital incision was made from the outer limit of the right frontal sinus down to the inner canthus. The lacrimal sac was dissected back from its bed, and the ethmoid labyrinth was exenterated. The front wall of the frontal sinus was completely removed, and in doing this it was found that this sinus was full of the same thick yellowish pus. When the sinus was completely exposed a large area, about the size of a five cent piece, was found in its superior lateral angle. It was covered with granulation tissue, with a small cone of dura easily seen in the middle of it. The granulations were sponged off. No

break was found in the dura. In removing the posterior wall of the frontal sinus much more bone was found to be soft and necrotic. An iodoform pack was put in. Two sutures were placed at the end of the incision at the inner canthus of the eye. The upper part of the wound was left open. The patient was sent to the ward with pressure dressing.

Thirty minutes after the operation he had a severe chill. A blood culture was taken at this time for typing for immunotransfusion. This was later found to be negative. The patient was well during the afternoon, and the next morning, when he was seen at 7 o'clock, he had no complaints. At 10 o'clock, when Dr. Poirier visited him, the patient had a pain in the right side of the chest.

Examination showed a few rales at the base of the right lung and a pleural rub. At this time a roentgenogram showed marked pneumonitis on both sides. The patient during the day was kept as quiet as possible with morphine. He took fluids well.

Dr. Means, of the medical service of the Massachusetts General Hospital, saw the patient and suggested merely supportive treatment and close watching. During the night, as the patient's breathing was more labored, he was placed in an oxygen tent. His color remained good. The next morning the breathing was more labored. The temperature was 103 F. The pulse rate was around 120 and the respiratory rate 35. A leukocyte count at the time showed 16,000 cells, with 82 per cent polymorphonuclears. Dr. Means again saw the patient and discussed with Dr. Lyons the feasibility of sulfanilamide treatment in view of the recovery of Staphylococcus albus from the pus.

Before the operation the patient had a spinal fluid pressure of 210. The differential blood count showed 8 polymorphonuclears and 8 lymphocytes.

Two days after operation the condition was much worse. The patient was comatose. Examination showed that the process had extended almost completely over the right side of the chest and affected most of the left lung. At 2:30 he died. Permission for postmortem examination was not obtained.

Dr. A. S. MacMillan: Postmortem roentgenograms showed complete consolidation of the right lung and behind the heart on the left side. Plates of the skull showed nothing remarkable.

Dr. F. M. Wattles: Cultures of the spinal fluid and two blood cultures were entirely negative. Hinton and Wassermann tests were reported negative.

DISCUSSION

Dr. G. H. Poirier: After operation the patient improved slightly, and my associates and I were never able to obtain any neurologic signs at all except for the spinal fluid findings. The cause of death was pneumonia.

Dr. H. P. Mosher: This case was brilliantly managed from the diagnostic and operative standpoint. I once made the remark that an exploration immediately or within a reasonable time for diagnosis was in order. It looks like an instance of postoperative pneumonia. Why it does not occur more often is a mystery to me.

Acute Suppurative Otitis Media on the Left, with Mastoiditis and Osteomyelitis of the Skull. Dr. G. H. Poirier, Dr. L. E. White, Dr. R. L. Goodale, Dr. P. Mysel, Dr. B. E. Lovesey, Dr. J. M. Converse and Dr. I. H. Blaisdell, with Roentgenologic Report by Dr. A. S. MacMillan.

Dr. B. E. Lovesey: A boy 15 years of age was first admitted to the hospital on Aug. 15, 1938, with a history of acute otitis media of five days' duration. During that time his temperature had ranged between 101 and 104 F. At the time of admission his temperature was 105 F. Cultures of the ear revealed a hemolytic streptococcus, and the patient was given adequate doses of sulfanilamide.

After this, his temperature subsided somewhat, and on August 25, ten days after admission, simple mastoidectomy was done on the right by Dr. R. L.

Goodale and Dr. J. M. Converse, as all blood cultures were positive, although the sulfanilamide content of the blood was 28 mg. per hundred cubic centimeters. At operation a large perisinal abscess was found, and the sinus itself was seen to be thickened and covered with necrotic granulations. The sinus was opened, and free bleeding from both ends was obtained. At this time also the patient presented symptoms of pulmonary infarction, which subsequently went on to the formation of an abscess which was drained surgically.

For a time after operation the patient responded fairly well, and then again he began to show a septic temperature. On September 22, the mastoidectomy wound was revised, and the internal jugular vein was ligated. During the next week the septicemia continued, and on September 29 the jugular bulb was explored. At this time there appeared also a reddened area on the right wrist, which was diagnosed by the surgical consultants as osteomyelitic. It was decided, however, that operative intervention for the osteomyelitis was inadvisable. During this period the patient had received large doses of sulfanilamide and had had a total of twenty-five blood transfusions.

He began to convalesce and was discharged from the hospital on October 20.

For the next two months he did well at home, but on December 19 he complained of postaural pain on the right side in the region of the operative defect, and for two weeks there had been a gradually increasing postaural swelling. He was readmitted to the hospital and roentgenograms were taken.

DR. A. S. MACMILLAN: They showed the defect of bone due to the mastoidectomy and an area of osteomyelitis posterior to it, with a large sequestrum at this point. An anterior-posterior view showed the same sequestrum in the occipital bone, within the area of osteomyelitis.

DR. B. E. LOVESEY: On December 22, a large area of osteomyelitic bone was removed by Drs. Mysel, Quincy and Drooker from the occiput, posterior to the mastoid defect, but the patient's condition on the operating table became so critical that the operator was forced to terminate the operation before its completion. Again the patient received several transfusions and apparently began to convalesce. However, during the last week of January 1939 he began to have repeated febrile attacks and complained occasionally of headache. There was considerable tenderness about the operative wound. Roentgenograms were again taken on February 10 and showed extension of the osteomyelitis.

DR. A. S. MACMILLAN: An area of osteomyelitis apparently extended up a region which the films taken before showed to be perfectly solid and in which they showed the blood vessels to be of normal size. In the later roentgenogram were shown blood vessels becoming more prominent and bone becoming definitely soft.

DR. B. E. LOVESEY: The following day further osteomyelitic bone was removed by Drs. Poirier and White, and an area of normal dura 3⅛ by 3⅝ inches (8 by 9 cm.) was uncovered. The patient withstood this operation fairly well but again received another transfusion, making the total number of blood transfusions which he had had thirty-one.

Follow-up roentgenograms taken on March 3 showed no further extension of the osteomyelitis.

DR. A. S. MACMILLAN: They showed the extensive removal of bone extending from the original site of operation to the posterior parietal and the occipital bone.

DR. B. E. LOVESEY: The patient began to improve, and the temperature returned to normal. However, on March 11, the right radius, which had apparently been quiescent for several months, flared up and become acutely tender and swollen. The following day the surgical consultant, Dr. Sarris, of the Massachusetts General Hospital, removed sequestrums and curetted the cavity. Since that time the patient's course has been uneventful, and all wounds are healing well. It might be of interest to know that a total of 5,198 grains (337 Gm.) of sulfanilamide were used in the treatment.

DISCUSSION

DR. H. P. MOSHER: It has been a long time since osteomyelitis has been associated with mastoidectomy. I can remember in the dim distant past 2 or 3 such cases. The second point, even more clearly evident now than before, is that, the condition of the patient permitting it, one should do as extensive an operation for osteomyelitis of the mastoid as one would for that of the frontal bone.

EDITOR'S NOTE: The patient had an uneventful course until Sept. 30, 1939, when he complained again of pain in the right elbow and forearm. He was referred to the osteomyelitis clinic of the Massachusetts General Hospital. A roentgenogram of the forearm and elbow showed findings consistent with infectious arthritis. The patient was given sulfanilamide, 15 grains (0.97 Gm.) daily, on the advice of Dr. Champ Lyons of the Massachusetts General Hospital. Under this treatment he has shown continued improvement, so that on December 6 sulfanilamide was omitted.

Chronic Suppurative Otitis Media on the Right, with Complications.
DR. H. P. CAHILL, DR. G. B. FRED and DR. J. C. DROOKER.

DR. J. C. DROOKER: A 14 year old girl came to this hospital on October 11. The history went back to three weeks before, when she had a severe head cold. Ten days before her admission to the hospital she had severe pain in the right ear, requiring her to stay in bed. Four days before admission the right ear began to discharge. The same day she had several attacks of chills and nonprojectile vomiting. Three days before admission she had an elevated temperature and vomited again. On admission, the patient had a temperature of 102 F. There were stiffness of the neck, bilateral Kernig signs and a positive Babinski sign. An immediate lumbar puncture showed a positive reaction to the Tobey-Ayer test on the right side, with 2,960 cells in the spinal fluid, 60 per cent of which were polymorphonuclears. The white blood cell count was 25,400, with 78 per cent polymorphonuclears. Examination showed a posterior perforation in the right membrana tympani. Little pus was seen. The patient had a periodic facial twitch every twenty-five seconds. The mother said it was a habit.

The past history revealed that at the age of 7 the patient had struck the right ear when she fell on a picket fence. A discharge from the ear followed this accident for a year and then subsided spontaneously.

Roentgenograms showed definite destruction of the right mastoid with no involvement of the petrous pyramid on the affected side.

A routine postaural incision was made with the patient under nitrogen monoxide–oxygen anesthesia, and when the cortex was uncovered thick yellow pus was obtained. The lateral sinus was uncovered and appeared to be collapsed. A thick fibrinous plastic exudate was found covering the cerebellar dura and the lateral sinus and extending further back, toward the occiput. For this reason a counterincision was made and the bone removed for 1½ inches (3.8 cm.) behind the lateral sinus. As far as the bone was removed the fibrinous exudate was present. The lateral sinus was packed off and incised and found to contain no blood when the packs were removed. A cannula was inserted in the upper end, toward the torcular Herophili, and free bleeding was obtained. There was no free bleeding from the lower end. The patient was returned to the ward and seemed to be getting along well.

A lumbar puncture was done the next day. The spinal fluid cell count was 540, with 44 per cent polymorphonuclear leukocytes.

On October 18, that is, seven days after operation, the patient complained of severe pain, not only on the diseased side but in the left ear as well. The left ear was examined and found to be perfectly normal. The stiffness of the neck by this time had subsided. The Kernig sign had disappeared. However, the

patient complained of extremely severe pain in the right side of the head and neck. Certain other signs had appeared: nystagmus to the opposite side and past pointing both sides, more on the right.

On October 19 the patient's pulse rate dropped to 70. A lumbar puncture on this day showed the presence of 67 per cent lymphocytes and an initial pressure of 100. Dr. James B. Ayer, of the neurologic service, saw the patient in consultation.

On the basis of the slow pulse, severe headache, increased blurring of the optic disks and condition of the spinal fluid as described, the decision was that the patient probably had a cerebellar abscess on the right. She was brought to the operating room. She had some respiratory distress and had Cheyne-Stokes breathing.

With the patient under nitrogen monoxide, oxygen and ether anesthesia, the dressing was taken down. It was noted that when the granulations in the mastoidectomy wound were cleaned the blood became black. The patient was found not to be breathing, and immediately one of the operators started to administer artificial respiration before the operation started.

The operation, which was done in two and one-half minutes, consisted of incising the medial wall of the right lateral sinus and inserting a Cushing needle to a depth of 4 cm. in a posterior-superior direction. Pus was immediately obtained. Because the patient was not breathing it was decided to remove all the pus present in the abscess, which amounted to 12 cc. The smear showed a hemolytic streptococcus. Subsequent cultures were negative. A rubber tube was inserted into the depth of the abscess, and after three minutes of artificial respiration the patient was breathing again.

That afternoon she had Cheyne-Stokes respiration again. In the evening her condition was poor; her color was poor and her respirations shallow. The drain was removed, and we looked for more walled-off pus. The finger was inserted for a depth of 3.5 cm., striking the cerebellar tentorium anteriorly. A large Mosher drain was inserted in the tract through the cerebral cortex and sutured in situ without anesthesia. The patient's condition improved somewhat and on the next day a small amount of pus drained through the Mosher basket. On the following day the respirations became so poor that it was decided to put the patient in a respirator. She was in the respirator for five days. For the next four days there was little drainage through the Mosher basket. The patient's condition was critical. Dr. Fred decided to remove the drain and put in a rubber tube. Another pocket was found, containing 2 cc. of pus. The condition now is rather poor, and she is kept breathing in the respirator. It is impossible for her to breathe when out of the respirator.

DISCUSSION

DR. L. A. SCHALL: This is another case of disastrous cerebellar abscess.

DR. G. B. FRED: At the time of operation we found no capsule, and the question in my mind at that time was whether to put in a drain or merely to let all the pus out and then withdraw the needle. I did put in a small rubber drain. The problem now is to get her to breathe, and the thing in the back of my mind is whether she could stand any more removal of bone from the outer edge of the foramen magnum so as to get operating space toward the medulla. The question is whether the pressure will go down with drainage and whether the medulla will function again.

DR. H. P. MOSHER: This is the first time it has been necessary to use a respirator in connection with abscess of the brain in this hospital.

Book Reviews

Medicine of the Ear. Edited by Edmund Prince Fowler Jr., M.D., Sc.D., Assistant Clinical Professor of Otolaryngology, College of Physicians and Surgeons, Columbia University; Assistant Surgeon, Manhattan Eye, Ear and Throat Hospital, New York. With a foreword by John Devereux Kernan, M.D., Professor of Otolaryngology, College of Physicians and Surgeons, Columbia University. Price, $12.00. Pp. 590, with 350 illustrations and 2 color plates. New York and Edinburgh: Thomas Nelson & Sons, 1939.

This newest addition to the Nelson Loose Leaf Series is a fit companion to "Surgery of the Ear," reviewed about a year ago in this journal. It has about 100 more pages and 50 more illustrations than the latter and is similar in binding and construction, with, of course, the loose leaf feature by which a subscriber, receiving additional material from time to time, keeps his volume up-to-date. All that was said about the Nelson "Surgery of the Ear" is applicable here, and Dr. Fowler deserves congratulations on his able work both as editor and as author of one of the important chapters.

As Dr. Kernan says in his foreword, it is necessary to correlate medicine of the ear with surgery of the ear, as well as with medicine and surgery of the nose and throat and with general medicine, and this has been well done in these two volumes. The present book gives all the essential anatomic, physiologic, etiologic and pathologic data and special tests for aural diseases and indicates when medical or surgical treatment becomes necessary.

The list of eighteen contributors is impressive. They were chosen from the foremost otologists in America, with the addition of two Europeans distinguished for their research work in the field of otology whose names are household words for every student of the ear. The bibliography attached to each chapter is extensive and most useful to the student. Stress is laid on pathologic changes and diagnosis, but treatment is not neglected.

The physiology of the organ of hearing is taken up in great detail and brought up to date, and much space is devoted to the newer diagnostic methods of examination of both the acoustic and the vestibular mechanisms, which of course are based on the physiologic observations and cannot be understood without a thorough knowledge of the latter. Modern methods for the management of the deafened person, including measures for improving his morale, are described, and the methods for prevention of deafness are outlined.

It seems to the reviewer that this work is excellent for the graduate student, the otolaryngologic hospital resident and the otologist but that much of it is too technical to appeal to the general practitioner. At the other extreme, however, there is much information that will appeal to the intelligent social worker and teacher and to other lay persons whose work is among school children and deafened adults.

The illustrations are excellent. If any portion should be singled out for criticism, it would be the inclusion of many pages of case histories. These are doubtless of interest to the student but seem just a little out of place in a work which should be classed as a textbook.

Les maladies de l'oesophage. By J. Terracol, Professeur a la Faculte de Medecine de Montpellier, with the collaboration of J. Baumel, S. Belinoff, P. Betoulieres, J. Delmas, G. Despons, F. G. Eeman, H. L. Guibert, P. Guns, M. H. Harant, F. Haslinger, P. Lamarque, Mounier-Kuhn, A. Peroni, M. Sargnon, J. Vialle, M. Wisner and M. Worms. Price, 220 francs. Cloth. Pp. 664, with 352 illustrations. Paris: Masson et Cie, 120 Boulevard Saint-Germain, 1938.

This comprehensive volume is an outstanding addition to the literature on the esophagus. It is well edited, and most of the chapters are written with a

minimum of verbosity. It is profusely illustrated with photographs and schematic and colored drawings, which are helpful, particularly to the reader whose knowledge of French is limited. The book is divided into four parts.

Professor Terracol and his collaborators are residents of continental Europe. Four of these men, however, Guns, Eeman, Peroni and Vialle, have been students in the Chevalier Jackson school of Broncho-Esophagology, and Dr. Jackson's teachings are well presented.

The first part of the book takes up anatomic considerations, both macroscopic and microscopic, with a chapter on the physiology of the esophagus. Methods of examination of the esophagus, roentgenography and esophagoscopy, are discussed in detail. The esophagoscopic technic and instrumentarium of Jackson, Moure, Guisez and Haslinger are presented. Haslinger himself is the author of a chapter. Exploration with the catheter and biopsy are briefly discussed. The chapter on accidents following esophagoscopy is well written and should be of great value in preventing such accidents.

The second part takes up pathologic conditions of the esophagus. Malformations, compression and neuromuscular abnormalities all are considered at length, with full notes on methods of treatment. Diverticula are discussed, and the surgical methods of treatment are well presented. Inflammatory lesions, peptic ulcer, tuberculosis, syphilis, diphtheria, esophageal complications of cutaneous infectious diseases, agranulocytosis and parasitic disease are included.

In the third part traumatic conditions of the esophagus are considered; rupture, accidental injury, perforations, abscess, corrosive burns, cicatricial stricture and tracheoesophageal fistulas are presented, with full description of methods of treatment. Continuous string retrograde dilation with flexible bougies for cicatricial stenosis is not mentioned.

The fourth part takes up tumors of the esophagus, benign and malignant. Radiologic procedures, both diagnostic and therapeutic, in the group of cases discussed in this part of the book are given full consideration. The chapter by Eeman on foreign bodies in the esophagus is particularly worthy of mention.

The last chapter is devoted to gastroscopic examination with the flexible tube of Schindler and is an excellent presentation of the subject.

The book is well written and well printed, and its scope and bibliographic reference make it a valuable reference work, despite the fact that it is not provided with an index.

News and Comment

AMERICAN BOARD OF OTOLARYNGOLOGY EXAMINATION

The American Board of Otolaryngology will hold an examination at the Manhattan Eye, Ear and Throat Hospital, New York, June 3, 4 and 5, 1940.

Directory of Otolaryngologic Societies *

FOREIGN

Collegium Oto-Rhino-Laryngologicum Amicitiæ Sacrum

President: Dr. Louis Ledoux, Brussels, Belgium.
Secretary: Prof. Dr. C. E. Benjamins, Verlengde Heereweg 143, Groningen, Netherlands.

Hungarian Otolaryngological Society

President: Dr. V. Zimányi, Zárda-u. 48, Budapest II.
Secretary: Dr. G. Kelemen, Reáltanoda-u., Budapest IV.

Sociedad Rioplatense de Oto-Rhino-Laringología (Argentine Section)

President: Dr. Raul Becco, B. Mitre 1690, Buenos Aires.
Secretary: Dr. Juan Manuel Tato, Santa Fé 1171, Buenos Aires.

Société Française d'Oto-Rhino-Laryngologie

Secretary: Dr. Henri Flurin, 19 Avenue Mac-Mahon, Paris, 17e.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. A. W. Proetz, 3720 Washington Blvd., St. Louis.
Secretary: Dr. Leroy A. Schall, 270 Commonwealth Ave., Boston.
Place: New York. Time: June 10-14, 1940.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Frank Brawley, 30 N. Michigan Ave., Chicago.
Executive Secretary: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Cleveland. Time: Oct. 6-11, 1940.

American Broncho-Esophagological Association

President: Dr. Lyman Richards, 319 Longwood Ave., Boston.
Secretary: Dr. Paul Holinger, 1150 N. State St., Chicago.
Place: New York. Time: June 5, 1940.

American Laryngological Association

President: Dr. James A. Babbitt, 1912 Spruce St., Philadelphia.
Secretary: Dr. Charles J. Imperatori, 108 E. 38th St., New York.
Place: Westchester Country Club, Rye, N. Y. Time: May 27-29, 1940.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Lee M. Hurd, 39 E. 50th St., New York.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester, N. Y.
Place: Waldorf-Astoria Hotel, New York. Time: June 6-8, 1940.

SECTIONS:

Eastern.—Chairman: Dr. John R. Simpson, Medical Arts Bldg., Pittsburgh.
Southern.—Chairman: Dr. Walter J. Bristow, Doctors Bldg., Columbia, S. C.
Middle.—Chairman: Dr. Sam E. Roberts, Professional Bldg., Kansas City, Mo.
Western.—Chairman: Dr. Pierre Viole, 1930 Wilshire Blvd., Los Angeles.

AMERICAN OTOLOGICAL SOCIETY

President: Dr. Horace Newhart, 527 Medical Arts Bldg., Minneapolis.
Secretary: Dr. Thomas J. Harris, 104 E. 40th St., New York.
Place: Westchester Country Club, Rye, N. Y. Time: May 30-31, 1940.